

THE NATURE OF WORKING MEMORY IN APHASIA

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Submitted to the faculty of the University Graduate School

in partial fulfillment of the requirements

for the degree

Doctor of Philosophy

in the Department of Speech and Hearing Sciences,

Indiana University

February 2007

Accepted by the Graduate Faculty, Indiana University, in partial fulfillment of the  
requirements for the degree of Doctor of Philosophy.

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February 16, 2007

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## **Dedication**

To Steve and Talia, for teaching me what matters.

## **Acknowledgements**

Special thanks to Laura Murray for her many hours spent teaching, listening, laughing, and editing; to Lyn Turkstra for stepping up in the midst of her own demanding schedule to be a dedicated mentor; to Judith Gierut, Raquel Anderson, and Olaf Sporns for their time, effort, and patience as committee members; to Julia Evans for her immense support in task and stimulus development; to Pamela Hadley and Matthew Rispoli for their insights and encouragement; and to Bonnie Lorenzen and Jennie Ostrander for their help in data collection. I am also deeply indebted to all of the participants who generously volunteered their time to be a part of this study.

Portions of this work were funded by:

- American Speech-Language-Hearing Foundation (ASHF) New Century Doctoral Scholarship
- Indiana University College of Arts and Sciences Dissertation Year Research Fellowship & Bernice Eastwood Covalt Memorial Scholarship
- National Institute on Deafness and Other Communication Disorders Grant RO1-DC03886 (PI: Laura Murray)

## ABSTRACT

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### THE NATURE OF WORKING MEMORY DEFICITS IN APHASIA

It is well known that many adults with aphasia demonstrate concomitant deficits in higher-level cognitive functions, including attention, executive function, and short-term and working memory. This has led to two premises: (a) the *domain-specific* hypothesis, in which aphasia is associated with additional cognitive deficits only to the extent that these are dependent upon language; and (b) the *domain-general* hypothesis, in which aphasia is associated with nonlinguistic cognitive impairments as a consequence of either overlapping anatomy or widespread cortical changes post-insult.

The purpose of this research was to disentangle these competing hypotheses with regards to working memory (WM) in adults with aphasia. Like other categories of cognitive impairment in this patient group, past research has identified but failed to elucidate WM impairments in aphasic language processing. Toward this end, 15 adults with left-hemisphere damage and aphasia (LHD) and 12 non-brain-damaged controls (NBD) completed a parametric WM task with systematic variation of psycholinguistic complexity (high-frequency, low-frequency, or non-nameable stimuli) and WM load (0-, 1-, and 2-back). Data were analyzed with respect to the differential impact of these variables within and across subjects and groups.

Whereas expected effects of word frequency were elicited in stimulus confrontation naming, LHD subjects were affected only minimally by frequency manipulations during the n-back task. Instead, these subjects demonstrated a significant performance decrement relative to controls with increasing WM load. Moreover, aphasia severity was moderately correlated with WM for non-nameable (i.e., more difficult) but not nameable stimuli. At the theoretical level, these results support a resource-based processing model in aphasia; at the neurobiological level, these findings are consistent with the proposition of widespread cortical connectivity changes irrespective of type or location of brain damage.

A secondary purpose of this study was to investigate the reliability of LHD performance on the n-back task, given the known performance variability associated with aphasia and the general dearth of reliability data for higher-level tasks. Results demonstrated that the n-back task is a reliable WM indicator over time for this population.

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# THE NATURE OF WORKING MEMORY DEFICITS IN APHASIA

## **I. Background and Rationale**

### **1. Comorbidity of aphasia and higher-level cognitive deficits**

Researchers over the last several decades have observed with increasing frequency that the language and communication problems observed in aphasia go beyond simply an impaired linguistic system and involve a complex mixture of cognitive deficits (Chapey, 2001; Chapey & Hallowell, 2001; Chapey, Riggio, & Morison, 1977; Helm-Estabrooks, 2001, 2002; Helm-Estabrooks, Bayles, Ramage, & Bryant, 1995; Murray, 2003; Murray, Baldwin, & Karcher, 2002; Murray & Ramage, 2000b; Purdy, 2002; Van Mourik, Verschaeve, Boon, Paquier, & Van Harskamp, 1992). This notion has received increased attention of late; indeed, Helm-Estabrooks (2002), referring to domains of cognition and their interaction with aphasia, concluded that “this is an area ripe for investigation as we rightfully move away from the conceptualization of language as being separate from cognition and accept that language is one aspect of cognition” (p. 184). Many patients with aphasia, but not all (Hamsher, 1998), demonstrate impairments in a variety of cognitive processes, including attention (Glosser & Goodglass, 1990; McNeil, Odell, & Tseng, 1991; Moineau, Dronkers, & Bates, 2005; Murray, 1999; Murray, Holland, & Beeson, 1997a; Robin & Rizzo, 1989; Tseng, McNeil, & Milenkovic, 1993), nonverbal and verbal fluency (Chapey et al., 1977; Glosser & Goodglass, 1990; Helm-Estabrooks,

2002; Jones-Gotman & Milner, 1977), short-term and working memory (Caspari, Parkinson, LaPointe, & Katz, 1998; Gutbrod, Cohen, Mager, & Meier, 1989; Valler, Corno, & Basso, 1992; Van Mourik et al., 1992; Ween, Verfaellie, & Alexander, 1996), cognitive flexibility (Papagno & Basso, 1996; Purdy, 2002), planning (Hjelmquist, 1989; Keil, 2003; Purdy, 2002; Shallice, 1982), and problem solving (Grigoriu & Mihailescu, 1979; Shallice & Burgess, 1991; Vilkki, 1988). These deficits can negatively impact functional communication (Hardin & Ramsberger, 2004; Kaplan, Gallagher, & Glosser, 1998; Keil, 2003), social, academic, and vocational outcomes (Murray, 2003; Ween et al., 1996), and reduce patients' ability to profit from treatment (Gallagher, 1994; Hinckley & Carr, 2001; Nusbaum & Small, 2001; Ramsberger, 1994; Van Harskamp & Visch-Brink, 1991; Van Mourik et al., 1992). As such, it would seem important to incorporate such findings into "descriptions, theories, treatment, and thought of aphasic syndromes" (Moineau et al., 2005, p.884), towards the twofold goal of formulating a more accurate or useful model of aphasia, and seeking best possible treatment options for patients.

Incorporating these variable and broad nonlinguistic deficits into a coherent definition of aphasia, however, has proved a daunting task. In fact, a consistent pattern of deficits or an identifiable relationship between linguistic and nonlinguistic deficits has yet to be found (Kaplan et al., 1998; Murray, 1999; Murray et al., 1997a; Tseng et al., 1993). For example, highly variable patterns of language deficits and nonlinguistic cognitive impairments in memory, attention, intelligence, and complex visual recognition have been reported for 173 left hemisphere patients subdivided by aphasia presence, type, and severity (Basso, Capitani, Luzzatti, & Spinnler, 1981), 13

patients with mild-moderate aphasia (Helm-Estabrooks, 2001), 17 patients with global aphasia (Van Mourik et al., 1992), and 58 patients with mixed aphasia types (Baldo et al., 2001).

Collectively, efforts to identify a relationship between linguistic and nonlinguistic deficits in adults with aphasia as well as other language-disordered populations (Ellis-Weismer, Evans, & Hesketh, 1999; Nation, Adams, Bowyer-Crane, & Snowling, 1999) have led to two primary hypotheses. The first, which we shall call a “general capacity hypothesis,” is that brain damage produces limitations in global attentional or WM resources (Basso, De Renzi, Faglioni, Scotti, & Spinnler, 1973; Haarmann, Just, & Carpenter, 1997; Wepman, 1972). Fueled by this standpoint, many investigators have posited alternate accounts for the language breakdown observed in individuals with aphasia. Common to these accounts is the presumption that deficits in nonlinguistic functions (e.g., attention allocation, processing capacity, working memory), imposed by either exogenous or endogenous conditions (Moineau et al., 2005), play an essential role in the generation (McNeil et al., 1991) or exacerbation (Ellis-Weismer et al., 1999; Murray, 1999; Murray & Kean, 2004) of language-based aphasic symptoms.

Such accounts, however, are not without dispute. In particular, one must bear in mind that aphasia affects a deeply ingrained linguistic system, and the direction of influence between language and nonlinguistic processing capacity is not entirely appreciated. For example, it has been argued that long-term language knowledge is inseparable from processing capacity (MacDonald & Christiansen, 2002; Mainela-Arnold & Evans, 2005), with bidirectional influences throughout the lifespan

(Dollaghan, Biber, & Campbell, 1993). According to these domain-specific accounts of language impairment, previous results showing processing capacity limitations in language-impaired populations (e.g., children with SLI) are simply an artifact of differences in the representational strength of long-term linguistic knowledge (e.g., poor recall of low-frequency words), moderating the need to impose additional, nonlinguistic processing constraints. From this perspective, the “domain-specific hypothesis” is that the primary language difficulties caused by left-hemisphere damage (or developmental language impairments) have a direct impact on other cognitive skills (Buckingham, 1985; De Renzi & Faglioni, 1965; Grigoriu & Mihailescu, 1979), especially given the covert verbal nature of many cognitive underpinnings (Luria, 1966; 1973).

Accordingly, although the literature supports the existence of cognitive processing impairments in aphasia, the dearth of theoretically driven investigations regarding the nature of these deficits disallows differentiation between domain-general and domain-specific accounts. Therefore, this study was designed to determine whether working memory deficits in aphasia can be better explained by a domain-general or domain-specific theory of cognitive processing. Exploration of working memory was chosen due to its increased tractability compared to broader constructs like executive function (Alexander & Stuss, 2006; Crawford, 1998; Keil, 2003; Salthouse, 2004). However, it is important to note that the working memory construct overlaps considerably with attention and short-term memory as well as with aspects of executive function (Awh, Vogel, & Oh, 2006; Murray & Kean, 2004).

A secondary purpose of this study was to investigate the reliability of subjects' performance on a parametric (n-back) working memory task. Although the reliability of this task has been reported for non-brain-damaged adults (Hockey & Geffen, 2004; Salthouse, Atkinson, & Berish, 2003), this information is not available for adults with aphasia. The lack of reliability information for cognitive assessment of adults with aphasia is all the more important given the well-known variability in these patients' day-to-day linguistic and cognitive performance (Tseng et al., 1993).

The following sections include a brief review of four interrelated cognitive deficit areas identified in patients with aphasia: short-term memory (STM), attention, executive function (EF), and working memory (WM), followed by a more detailed review of the WM construct. Dichotomous and parallel issues will be presented with respect to: 1) the nature of WM deficits in patients with aphasia, and 2) the nature of WM in the normally functioning cognitive system. Although these areas of research are historically discrete (e.g., distinct laboratories, methodology, and subject groups), this review will show that the general theme identified in each camp can be narrowed down to the classic debate between theories of domain specificity and domain generality in cognitive processing.

### ***1.1 Aphasia and short-term memory***

Although the terms “short-term memory” (STM) and “working memory” (WM) are often used interchangeably in the literature, a critical distinction exists: Whereas STM refers to an individual's ability to store or maintain information over a certain (limited) time period, WM refers to an ability to hold information *while*

*manipulating or integrating other information*, in the service of some cognitive goal (Jarrold & Towse, 2006; Kane & Engle, 2002). Given what is known about STM anatomy and processing operations, the integrity of STM in healthy adults is largely thought to reflect domain-specific storage operations, such as capacity for maintaining a phonological code (Baddeley, Thomson, & Buchanan, 1975). Accordingly, the relationship between aphasia and STM is historically assumed to reflect domain-specific, language-related factors.

Martin and colleagues (Freedman & Martin, 2001; Martin & Feher, 1990) have closely examined the relationship between STM span and language processing in patients with aphasia. For example, Martin (1987) found that many individuals with aphasia failed to show the phonological similarity and word length effects that are often taken as evidence of covert verbal rehearsal in the phonological loop (Baddeley, 2000, 2002; Baddeley & Hitch, 1994), supporting the hypothesis that aphasia reflects in part decreased retention of phonemic sequences (Martin, Breedin, & Damian, 1999). Furthermore, as predicted by other researchers (Caplan & Waters, 1995), Martin and Feher (1990) found that reduced memory span in their subjects with aphasia did not affect comprehension of syntactically complex sentences, but rather affected comprehension of sentences with increased numbers of content words. On the basis of these and similar results, Martin and colleagues (Freedman & Martin, 2001; Martin, Lesch, & Bartha, 1999) have contended that multiple linguistic codes (e.g., dissociable phonological and semantic components) support verbal short-term memory, and that these separable retention capacities may be selectively affected by brain damage, independently of their respective processing roles.

Although other researchers have supported the role of language in STM information acquisition, evidence for separable retention versus processing capacities has been mixed. For example, Ween et al. (1996) administered a variety of short- and long-term memory measures to 16 individuals with mild aphasia, and found that the presence of aphasia affected acquisition of information into both short- and long-term memory, but that these effects could be explained entirely by linguistic processing deficits, with phonological impairment affecting the former and lexicosemantic impairment affecting the latter (i.e., self-organized encoding of word lists).

Interestingly, the few studies which have included both verbal and nonverbal STM tasks have failed to support a pivotal role for aphasic language impairments in STM. For example, Burgio and Basso (1997) found that individuals with aphasia ( $n = 61$  acute and 17 chronic patients) were impaired on verbal and spatial short- and long-term memory tasks, especially in the acute phase. Although Burgio and Basso failed to find a difference between the performance of patients with anterior versus posterior lesions, an earlier but smaller study ( $n = 4$  per aphasia group and 4 NBD controls) demonstrated nonverbal visual memory deficits in patients with Broca's but not Wernicke's aphasia, along with expected verbal memory deficits for both patient groups (Ostergaard & Meudell, 1984). These authors suggested that language deficits predicted memory deficits in Wernicke's aphasia, whereas the nonverbal memory deficits in Broca's aphasia indicated a wider-ranging mnemonic deficit, perhaps due to "a general inability to appreciate structure inherent in any material" (p.12). Other authors have similarly suggested that individuals with aphasia have a domain-general impairment in extracting inherent stimulus structure (Basso, Capitani, Luzzatti,



Spinnler, & Zanobio, 1985; Gainotti, Carlomagno, Craca, & Silveri, 1986; Nicholas, 1999; Noppeney & Wallesch, 2000; Nusbaum & Small, 2001; Wayland & Taplin, 1982, 1985). For example, Dominey, Hoen, Blanc, and Lelekov-Boissard (2003) described a recurrent network model that explicitly established functional relationships between sequential cognition, i.e., “the capabilities to extract and utilize the sequential structure of perceptual and motor events in the world in an adaptive and pragmatic manner” (p. 208), and language. Results of model simulations led Dominey et al. to two specific predictions: First, brain damage which negatively affects syntactic comprehension (e.g., agrammatic or nonfluent aphasia) should also affect analogous, non-linguistic or abstract cognitive sequences; second, NBD subjects’ neuroimaging data should reveal common neurophysiological substrates for such tasks (e.g., the LAN (left anterior negativity) language-related ERP effect). Dominey et al.’s results supported both predictions, leading the authors to conclude that, although language is clearly dependent on dedicated neurophysiological processes to some extent, “there is also a substantial functional and neurophysiological overlap between aspects of language processing and non-linguistic sequential cognition” (p. 222). This is especially relevant given that classic theories of the phonological loop, the verbal STM component of WM (Baddeley, 1986), maintain the most useful aspect of the loop is its capacity for storing serial order of verbally recoded materials (Baddeley, 2000).

In sum, the literature generally suggests domain-specific STM problems associated with aphasia, which stem either directly from language processing impairments or from functionally separable, code-specific retention capacities.

However, the domain-specificity of these memory problems may be due in part to a failure of many of these studies to include both non-verbal as well as verbal memory tasks. Thus, it is also possible that individuals with aphasia sustain damage to both linguistic and non-linguistic sequential processing related to STM.

### ***1.2 Aphasia and attention***

Attention has been functionally defined in a number of ways (see reviews in Murray, 1999; Posner, 1994), but a general model based on resource allocation (Kahneman, 1973) has proven particularly useful in accounting for deficient performance by individuals with brain damage in a variety of situations. As applied to aphasia, a large number of studies have demonstrated that the language-specific deficits of patients with aphasia co-vary with the demands placed on cognitive resources (Blackwell & Bates, 1995; McNeil, Hula, Matthews, Doyle, & Fossett, 2004; Moineau et al., 2005; Murray, 1999; Silkes, McNeil, & Drton, 2004), supporting a resource-driven perspective in which aphasia is associated with limited attentional resources, misallocation of resources, or both (Miyake, Carpenter, & Just, 1994, 1995; Murray, 1999). The appeal of resource theories lies in their “potential for explaining the perseverations, intrusions, confusions, and shifts of linguistic items in aphasic speech production and perception without appealing to the deterministic linguistic models” (Tseng et al., 1993, p. 292). Although some authors have emphasized the effect of attentional deficits on language performance (Robin & Rizzo, 1989), other researchers have demonstrated generalized deficits in patients that cut across verbal and nonverbal domains, indicating that these cognitive deficits are

likely independent of linguistic skills (Lang, 1989; Laures, Odell, & Coe, 2003; Shisler, 2005). A possible neurophysiological explanation of these trends was given by Petry and colleagues (Petry, Crosson, Gonzalez-Rothi, Bauer, & Schauer, 1994), who found selective attention deficits (i.e., slower responding to right hemispace) in 13 patients with aphasia (compared with 13 healthy controls), which correlated with impaired performance on six of seven language measures. These authors suggested that the ability to adequately employ left-hemisphere processing mechanisms during attentional tasks demonstrates the availability of intact processes with which to reconstruct a functional language system following left-hemisphere brain damage.

LaPointe and Erickson (1991) utilized a dual-task paradigm to monitor the performance of six adults with aphasia, and six age- and gender-matched controls, on an auditory vigilance task (monitoring a stream of words for one in particular) presented in isolation and in tandem with a simple card-sorting task. Whereas the performance of controls and subjects with aphasia was nearly identical when the vigilance task was performed alone, the subjects with aphasia experienced a significant dual task decrement compared to the control group. These results were replicated and extended by Erickson, Goldfinger, and LaPointe (1996) for 10 adults with aphasia and 10 control subjects using a nonlinguistic auditory vigilance task (tone detection). The researchers interpreted these data as providing evidence of attentional allocation problems in aphasia, “in conjunction with, or superimposed on, [the] linguistic deficit” (LaPointe & Erickson, 1991, p. 518), irrespective of the nature of the stimulus (linguistic or non-linguistic), and especially in the presence of competing stimuli.

Laures et al. (2003) suggested that the previously reported lack of vigilance differences between aphasia and control groups (Erickson et al., 1996; L. L. LaPointe & Erickson, 1991; Petry et al., 1994) stemmed from failure to employ valid vigilance tasks, traditionally defined as lasting at least 30 minutes (Davies & Parasuraman, 1982). In contrast, Laures et al. observed deficient overall arousal and vigilance in their sample of 10 participants with aphasia, compared with 10 NBD controls, across simple, 30-minute linguistic and nonlinguistic auditory vigilance tasks, indicating that more primitive deficits in sustained attention might underlie the poor performance of patients with aphasia in dual-task studies (McNeil et al., 1991). Interestingly, these authors found that physiological measures of blood pressure and cortisol levels dovetailed with their behavioral data, with a lower mean systolic and diastolic blood pressure (after correcting for anti-hypertensive medication use), and a higher level of cortisol secretion, for the aphasic group compared to the control group at baseline, and only modest increases in blood pressure (but not cortisol levels) during experimental periods. Laures et al. interpreted these data as reflecting a “failure of the cardiovascular and pituitary-adrenal systems of the aphasic participants to react to the challenge of performance” (p. 1145), potentially due to poor self-monitoring of performance accuracy.

Tseng et al. (1993) examined 9 left-hemisphere-damaged adults with aphasia, compared to 18 healthy controls, in a dual-task paradigm which required subjects to engage in phonemic monitoring, semantic judgment, or both tasks, with manipulation of target probabilities across tasks. Although control subjects were able to exploit both implicit as well as explicitly given target probabilities to decrease task reaction

time (RT), subjects with aphasia did not show any sensitivity to probability in any of the tasks. Tseng et al. concluded that their subjects with aphasia displayed deficient attention allocation processes, due to a failure to appropriately evaluate task demands, slowed attentional mobilization, or both. A similar interpretation of dual task data was given by Murray and colleagues (Murray et al., 1997a; Murray, Holland, & Beeson, 1997b), who examined the performance of 16 individuals with mild aphasia (8 individuals with frontal and 8 with posterior lesions) and 8 healthy controls during lexical and semantic listening tasks (1997a) and a grammaticality judgment task (1997b), presented under isolation, focused attention, and divided attention conditions. Their subjects with aphasia demonstrated deficits in inhibiting processing of competing stimuli, allocating attention across tasks, and judging task demands compared to controls; moreover, these impairments appeared independent of concomitant language deficits, according to experimental correlational analyses. That Murray et al. (1997a) found no differences between the anterior and posterior patients suggested complex interactions between frontal and posterior brain regions in allocating attentional resources. Like Tseng et al., Murray (1999) suggested that such difficulties in appropriately allocating cognitive resources might represent a failure of the individuals with aphasia to judge appropriately either task demands, performance capabilities, or both.

### ***1.3. Aphasia and executive function***

The association of aphasia with executive function (EF) derives from early aphasia models, which emphasized the association of the disorder with more general

cognitive deficits (Edwards, Ellams, & Thompson, 1976; Larrabee, 1986). Kurt Goldstein (Goldstein, 1948; Goldstein & Scheerer, 1941; Noppeney & Wallesch, 2000), for example, considered aphasia to represent a disorder of symbolic thinking or “abstract attitude,” in which affected individuals were unable to actively structure or categorize input. Goldstein’s abstract-concrete continuum was reflected in Chapey et al.’s (1977) divergent-convergent interpretation of aphasia as a divergent semantic impairment. To Chapey et al., “it is the divergent component of propositional, spontaneous, and abstract language” that is most impaired in aphasia, which is most apparent when “communication requires formulating and searching for a variety of new ideas and relationships, which produce a number and a variety of different kinds of categories of response, and extend the boundaries of what is already known” (p. 293).

Luria’s (1961; 1966; 1973) theoretical stance on the control of perception, planning, and intentionality by verbal behavior strongly influenced many early studies of aphasia and EF (Denckla, 1996). For example, Hjelmquist (1989) administered an object-sorting test to 30 subjects with aphasia, 17 NBD controls, and 13 right-hemisphere-damaged subjects. The task required subjects to sort real objects in as many different ways as possible (e.g., by function, color, or form), and Hjelmquist identified and measured four task variables, including “quality” (i.e., ability to categorize), “planning” or organizational strategies, “shifting” across category types, and “stability” within abstract categories. Compared to the other groups, the subjects with aphasia scored lower across all task variables, with fluent participants scoring slightly lower than nonfluent participants in “quality,” but not the

other dimensions. Hjelmquist concluded that these subjects' deficient planning skills were a product of their language deficits in that "although these patients usually use words as labels, these words do not function to control their behavior effectively" (p. 253). The inability of some patients with aphasia to control their behavior via language was likewise noted by Papagno and Basso (1996) in their examination of perseverative behavior in aphasia. A similar observation was made by Larabee (1986), who examined WAIS Verbal and Performance IQ (VIQ and PIQ, respectively) in 18 subjects with aphasia compared to 19 subjects with right-hemisphere damage. Larabee found that PIQ scores correlated significantly with language impairment in the left-hemisphere patients, reflecting the significant influence of language on purportedly nonverbal tasks composing the PIQ. These results were corroborated by Borod, Carper, and Goodglass (1982), who found that auditory comprehension abilities significantly influenced WAIS PIQ scores in 98 patients with aphasia.

The highly variable relationship between aphasia and EF across individual studies is highlighted by the results of studies exploring the relationship between aphasia and performance on the *Raven's Progressive Matrices* (RPM, J. Raven, Court, & Raven, 1985; 1999) or *Raven's Colored Progressive Matrices* (RCPM, J. Raven, Raven, & Court, 1998). A frequently utilized cognitive measure in aphasia due to its purportedly low verbal demands, these tests are designed to measure intelligence, logical reasoning, mental flexibility, spatial relations, and response selection (Hamsher, 1998; Keil & Kaszniak, 2002). Edwards et al. (1976) administered the RCPM to 62 patients with aphasia and found a statistically

significant but moderate correlation between RCPM performance and language skills as measured by the *Minnesota Test for Differential Diagnosis in Aphasia* (MTDDA, Schuell, 1965). Although “any supposition that intelligence and language skills are unrelated is denied by these findings,” they noted that “it is hard to see what this correlation reveals about a causal link between the two” (p. 90). A more detailed analysis revealed only weak relationships between simpler language tasks and RCPM scores, with progressively higher correlations between RCPM and more complex language tasks (e.g., writing). Edwards et al. concluded that, although language may have supported RCPM performance to some degree in their subjects, their results more likely reflected the reverse direction of causation: Intellectual ability supported language competence. Bailey, Powell, and Clark (1981), on the other hand, found that although RPM scores correlated negatively with initial severity and positively with aphasia recovery in 134 cases, a significant change in MTDDA scores did not coincide with RPM changes. Baldo et al. (2001) obtained a significant correlation between aphasia severity and RCPM performance in 58 patients with aphasia. Although the main effect of aphasia type (Wernicke’s, Broca’s, conduction, and anomic) on RCPM scores failed to reach significance, Baldo et al. reported that Wernicke’s, Broca’s, and conduction aphasia patients all performed significantly more poorly on this measure compared to anomic as well as non-aphasic chronic stroke patients. A more recent study by this group (Baldo et al., 2004) similarly found a significant relationship between severity of the language impairment ( $n = 41$  LHD patients) and RCPM scores. In contrast, an earlier study by Kertesz and McCabe (1975) failed to reveal a correlation between RCPM performance and overall aphasia



severity as measured by the *Western Aphasia Battery* (Kertesz, 1982) in 111 cases. Instead, in their study, patients' auditory comprehension predicted RCPM scores: Patients with poor comprehension did more poorly on the RCPM. Basso et al. (1973), however, failed to obtain a significant correlation between auditory comprehension scores on the *Token Test* (De Renzi & Vignolo, 1962) and the RCPM in 55 patients with aphasia, although they noted that patients with aphasia did more poorly than non-aphasic, left-hemisphere-damaged patients on the RCPM even after correction for lesion size and time post-onset. De Renzi and Faglioni (1965) found no differences between left aphasic ( $n = 58$ ), left non-aphasic ( $n = 40$ ), and right hemisphere groups ( $n = 68$ ) on the RCPM. Noting, however, that their right hemisphere patients presented with a more severe degree of cerebral damage, they concluded that "the left hemisphere is crucial for all intellectual tasks, verbal as well as non-verbal" (p. 429). Gainotti, Caltagirone, and Miceli (1977), on the other hand, after examining the performance of 179 left and 173 right brain-damaged patients on the RCPM, contended that both the right and left hemispheres were critical for visual-spatial and linguistic intelligence, respectively. Finally, Grigoriu and Mihailescu (1979) performed a more detailed examination of cognitive strategies employed by patients with aphasia compared to right-hemisphere patients and normal controls on the RPM. These researchers found that individuals with aphasia relied on more "primitive" strategies compared to the other two groups, resulting in normal performance for simpler items and significantly poorer performance for more difficult items, and concluded that their findings demonstrated the "control exerted by the verbal symbol upon the logic process" (p. 301). In sum, studies of the relationship

between aphasia and RPM or RCPM performance have found generally depressed scores in adults with aphasia compared to NBD controls as well as to adults with other types of brain damage, with mixed findings as to whether these results relate directly to the severity of the language impairment (Baldo et al., 2001; Edwards et al., 1976), auditory comprehension deficits (Basso et al., 1973; Kertesz & McCabe, 1975), aphasia recovery (Bailey et al., 1981), or general cognitive processing strategies (De Renzi & Faglioni, 1965; Gainotti et al., 1977; Grigoriu & Mihailescu, 1979).

In one of the first aphasia studies to address specifically the concept of EF, Shallice (1982) tested 61 patients with unilateral lesions (divided into four groups by lesions location: right/left, anterior/posterior) and 20 healthy controls using the Tower of London task (TOL), which assesses planning, forethought, and sustained attention (Keil & Kaszniak, 2002; Lezak, 1993), or inhibition (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). They found that the left anterior patients were most impaired on this task, with slow, inefficient performance and an inability to complete more complex problems. Calling this a “specific impairment in planning,” Shallice (1982) argued that the deficient performance of left anterior patients was not attributable to verbal or visual aphasia-related STM deficits, given the relatively unimpaired performance of left posterior patients. Nor could these results be explained by verbal mediation or the “regulating function of speech” (Luria, 1961; 1966; 1973), given the lack of an articulatory suppression effect with NBD subjects (i.e., repeating “ABCDEFGH” continuously while completing the TOL; cf. Baddeley, 1986). Instead, Shallice postulated the existence of a Supervisory Attentional System

(SAS), a “general programming system” within the left frontal lobe necessary for successful completion of non-routine tasks, and which could be impaired separately from or in addition to language following a neurological insult.

Whereas the SAS has served as an important impetus for many subsequent studies, other researchers contend that EF likely is a product of bilateral frontal lobe connections. For example, Glosser and Goodglass (1990) administered four nonverbal, experimental EF tests to 22 aphasic patients, 19 right-hemisphere patients, and 49 NBD controls. Their tasks included the following: (a) Nonverbal Continuous Performance Test, a visual vigilance task that measures sustained and selective attention, (b) Graphic Pattern Generation and Sequence Generation Tasks, which measure fluency or divergent thinking, and (c) Tower of Hanoi, a variant of the TOL that similarly assesses planning, forethought, and sustained attention, or possibly inhibition. Glosser and Goodglass reported dissimilar performance across subjects with aphasia according to lesion location, with frontal-lobe aphasic patients performing more poorly than those with nonfrontal lesions. In contrast to Shallice (1982), a similar performance pattern was obtained for right-hemisphere patients, leading Glosser and Goodglass (1990) to conclude that “lesions in the frontal lobes in either hemisphere contributed equally to impairments in executive control” (p. 498), even after controlling for lesion size. They further specified that EF dysfunction in their subjects with aphasia appeared entirely separable from language skills. This conclusion was supported and further explicated by Baldo and Dronkers (1999), who likewise noted that similar-looking deficits in EF tasks could be produced by a

deficient phonological store in patients with temporoparietal lesions and conduction aphasia, or by deficient attention allocation in patients with prefrontal lesions.

The complex interactions between phonological WM, attention, and EF were emphasized by Dunbar and Sussman (1995), who administered the *Wisconsin Card Sorting Test* (WCST) to healthy subjects divided into three groups: a control group who completed only the WCST and two experimental groups who completed the WCST concurrently with either an executive task (adding numbers progressively) or a phonological WM task (repeating numbers). The WCST is a classic test of frontal lobe function that assesses WM, reasoning, abstract concept formation, set shifting and maintenance, and feedback utilization (Dehaene & Changeux, 1991; Dugbartey, Rosenbaum, Sanchez, & Townes, 1999; Konishi et al., 2002; Spreen & Strauss, 1998). Contrary to their expectations, Dunbar and Sussman (1995) found that their subjects in the phonological rather than executive condition looked most like frontal patients on the WCST, with fewer categories obtained and more perseverative errors. These results were replicated in a second experiment using “purer” secondary tasks to tap EF and phonological STM (dynamic attention allocation and articulatory suppression, respectively). In a third experiment, Dunbar and Sussman administered the WCST to a patient diagnosed with conduction aphasia following a temporoparietal lesion who demonstrated a “pure case of an articulatory rehearsal deficit” (p. 296). That this patient obtained zero categories and demonstrated severe perseveration on the WCST supported their conclusion that impaired phonological memory, in the absence of a frontal lesion, can lead to executive dysfunction. Finally, in fourth and fifth experiments, Dunbar and Sussman found that patients with frontal

lobe lesions (of unspecified lateralization) were unable to complete tasks that demanded dynamic attention allocation. They concluded that frontal, executive deficits were actually a product of a complex set of interactions between “lower-level” cognitive processes, in which deficient attention allocation prevented adequate utilization of the phonological store, leading to EF dysfunction. These results were supported by Baldo et al. (2004), who found a significant correlation between severity of the language impairment in 41 subjects with aphasia and performance on the WCST, as well as significant impairments in WCST performance for healthy control subjects under the condition of articulatory suppression.

Purdy (2002) gave 15 subjects with aphasia four nonverbal EF tasks, including the Porteus Maze (PM), WCST, TOL, and TOH. The subjects with aphasia performed with equal accuracy to a control NBD group on the TOL and PM, but not the WCST or TOH. However, the subjects with aphasia displayed deficits in speed and efficiency across all four tasks, which Purdy interpreted as reflecting deficient cognitive flexibility and planning.

It is possible that the formal language tests employed to compare language and EF in previous studies did not adequately capture the linguistic or communicative impact of EF dysfunction in individuals with aphasia. Indeed, a more consistent relationship between EF and language in aphasia has been established when the latter is measured with respect to functional communication or treatment outcomes (Ramsberger, 2005). Keil (2003), although noting that patients with aphasia did more poorly than controls on “some tests” of EF including the RCPM, TOH, PM, and the Errands Test (an experimental measure of spatial planning), failed to find a

relationship between aphasia severity (as measured by the WAB), auditory comprehension, or ideomotor praxis and EF in 25 patients with aphasia compared to 7 patients with frontal lobe damage and 25 NBD controls. Keil did, however, obtain a significant relationship between scores on the *ASHA Functional Assessment of Communication Skills* (ASHA FACS, Frattali, Thompson, Holland, Wohl, & Ferketic, 1995) and performance on the Errands and PM tasks in her participants with aphasia. She concluded that cognitive impairments of planning and strategy use impact functional communication skills in aphasic patients. Likewise, Murray and Ramage (2000a; 2000b), who similarly found no apparent association between executive dysfunction and aphasia type or severity across a battery of EF and aphasia tests given to seven individuals with aphasia (including the *Behavioral Assessment of Dysexecutive Syndrome* (BADS), CLOX, WCST, Stroop test, Trails, *Test of Nonverbal Intelligence* (TONI), and RCPM), noted an apparent association between EF and aphasia treatment outcomes in their patients. Finally, Goldenberg, Dettmers, Grothe, and Spatt (1994) found a positive relationship between explicit nonverbal recall and success of intensive language therapy. Although these researchers failed to find a significant correlation between EF (as measured by sorting, association learning, and design fluency) and therapy outcomes, it has been argued that “explicit learning” or deliberate memorization is a key task of the central executive (Gutbrod et al., 1989).

In sum, patients with aphasia perform more poorly than controls across a variety of tasks purported to measure EF, a general result that appears unrelated to the language impairment per se (Murray & Ramage, 2000a, 2000b), although a deficient

phonological store (possibly related to a more general, attentional impairment; Dunbar & Sussman, 1995), may play a role. Moreover, there have been mixed reports as to whether these EF deficits reflect simply the known EF problems accompanying prefrontal lesions (Baldo & Dronkers, 1999; Glosser & Goodglass, 1990; Shallice, 1982), or whether these deficits are universal in aphasia (Purdy, 2002). Finally, a more consistent relationship between EF and language function has been reported when the latter is measured with respect to the role of language in everyday (complex) tasks or treatment outcomes (Keil, 2003; Ramsberger, 2005).

#### ***1.4 Aphasia and working memory***

An intact WM system is crucial for language processing (Aboitiz, Garcia, Bosman, & Brunetti, 2006; Caplan & Waters, 1999; Wright & Shisler, 2005). Therefore, a number of researchers have explored the integrity of WM in adults with aphasia. Two primary approaches have emerged. The first, reflecting paradigms such as Just and Carpenter's (1992) WM model for language, assumes that individuals with aphasia might experience language problems to the extent that they have WM impairments *specific to language* (Caplan & Waters, 1999; Caspari et al., 1998). This approach is compatible with both single- (Just & Carpenter, 1992; Miyake et al., 1994) and dual- resource WM theories (e.g., the *Separate Language Interpretation Resource* theory; Caplan & Waters, 1999). The second approach, reflecting theoretical models in which WM includes an executive component as well as domain-specific processing capabilities (Baddeley, 1986), has capitalized on evidence of domain-general, executive-type WM impairments in aphasia (Baldo & Dronkers,

1999; Beeson, Bayles, & Kaszniak, 1993; Downey et al., 2003), or evidence of improved language performance in individuals with aphasia given domain-general WM support (Linebarger, McCall, Virata, & Berndt, 2007).

Towards the first approach, Caplan, Waters, and colleagues (Caplan & Waters, 1995, 1999; Rochon, Waters, & Caplan, 2000) have studied WM capacity in individuals with aphasia to support their conceptualization of WM for language as modular and domain-specific. To these authors, the verbal WM system used for syntactic comprehension and the extraction of semantic and syntactic features from linguistic signals, i.e., “interpretive processing,” is entirely separate from the WM system used for other verbally mediated tasks, such as reasoning, planning, or storing semantic information, i.e., “post-interpretive processing” (Caplan & Waters, 1999, p. 78). The basis of their claims stems from research demonstrating that the effects of syntactic complexity versus concurrent memory load appear separable across clinical groups. For example, adults with aphasia failed to show exacerbation of syntactic processing deficits under a concurrent memory load (Caplan & Waters, 1996), whereas patients with dementia of the Alzheimer’s type (DAT) and Parkinson’s disease (PD) showed intact syntactic processing and severely impaired memory spans (Caplan & Waters, 1995; Rochon, Waters, & Caplan, 1994). These effects support Caplan and Waters’ (1999) contention that “working memory capacity, as measured on a task that emphasizes controlled, conscious manipulation of verbal information, will not correlate with processing efficiency for any components of the interpretation process” (p. 93), because the two types of processing are controlled by entirely



different cognitive systems (but see, e.g., Bates, Dick, & Wulfeck, 1999; Engle, 2002, for domain-general explanations of these results).

Several other researchers have endorsed specific, language-related WM impairments in aphasia, but have conceptualized such impairments as reflecting a single, low-capacity linguistic resource pool, rather than distinguishing between interpretive versus post-interpretive aspects of language. Miyake et al. (1994; 1995), for example, proposed that comprehension deficits in aphasia were the product of reduced WM capacity for language: specifically, a reduction in the activation resources needed to process incoming language and retain intermediate products of this processing. Toward this end, they reported a series of experiments with non-brain-damaged adults, divided into low- and high-span groups using Daneman and Carpenter's (1980) reading span task. Miyake et al. (1995) replicated the comprehension patterns of patients with aphasia (Caplan, Baker, & Dehaut, 1985) by inducing temporal constraints during a reading task (i.e., rapid serial visual presentation, RSVP). That is, subjects with low WM spans performed more poorly on complex sentence types than subjects with higher WM spans, and these low-WM subjects also performed more poorly at faster RSVP rates. That normal adults showed similar patterns to adults with aphasia led Miyake et al. to conclude that "at least some of the aphasic performance characteristically attributed to selective impairments may instead be attributable to individual variations in performance that are already present among normal adults, manifesting themselves in an exaggerated form as a result of brain damage" (p. 669; although see Caplan & Waters, 1999, for an alternate interpretation). They endorsed a normal-to-aphasia continuum of WM resources for

language comprehension, based on resource constraints as well as resource allocation strategies that are common to both groups of individuals.

A separate line of research that has been pursued involves administering WM span tasks, similar to Daneman and Carpenter's (1980) reading span task, directly to individuals with aphasia and correlating WM span performance with aspects of linguistic skills. Such studies have simplified the reading span task (Caspari et al., 1998), changed task input and response modalities, or both (Tompkins, Bloise, Timko, & Baumgaertner, 1994). Tompkins et al. administered a modified, auditory version of the reading span task to 75 individuals, including 25 right-hemisphere-damaged (RHD), 25 left-hemisphere-damaged (LHD), and 25 NBD subjects. As expected, the NBD group performed much better than both clinical groups; moreover, the LHD and RHD groups did not differ from one another in estimated WM capacity. Building on the logic of Just and Carpenter (1992), Tompkins et al. predicted that WM and discourse comprehension would be associated, but only as processing demands approached subjects' capacity; therefore, no meaningful associations between WM and comprehension were predicted for NBD subjects. Consistent with their predictions, correlations between WM and discourse comprehension for both RHD and LHD patients increased in magnitude as the demands of the comprehension task increased. The authors additionally observed that the comprehension skills of the LHD subjects predicted their estimated WM capacity; moreover, as in NBD populations (e.g., Daneman & Carpenter, 1983; Engle, 2002), WM scores for the aphasic group correlated with estimated IQ. Tompkins et al. recommended that the association between WM and other computationally demanding tasks be investigated

in brain-damaged individuals, “to ascertain the extent to which ‘general factors’ can account for seemingly specific deficits after brain damage” (p. 910). Likewise, Caspari et al. (1998) administered a simplified version of the reading span task to 23 individuals spanning a wide range of aphasia types and severity levels. Consistent with Daneman and Carpenter’s results with NBD subjects, they found strong, positive correlations between WM task scores and measures of language comprehension (both written and spoken) across their subjects with aphasia, and concluded that “the ability of aphasic individuals to comprehend language is predictable from their working memory capacities” (p. 205). Finally, Wright, Newhoff, Downey, and Austermann (2003) similarly found significant associations between LHD subjects’ WM capacity, measured using Tompkins et al.’s (1994) span task, and discourse comprehension skills, and concluded that resource-demanding language processing tasks exceeded WM capacity limits in adults with aphasia.

Although all of these authors modified the reading span task to accommodate the needs of their subjects with aphasia (e.g., reduced sentence length and complexity, requiring only a recognition response versus free recall), Caspari et al. (1998) noted that “it is well documented that aphasic individuals are handicapped at understanding even short active sentences, especially those in which meaning is determined by word order... therefore, even though the sentences were simplified, the task entailed both processing and storage, making it a complex task” (p. 210). That this task relied heavily on language processing resources cannot be ruled out as a primary factor in the authors’ obtained correlations between their WM and language measures (see MacDonald & Christiansen, 2002). Therefore, these collective results support a

linguistically constrained verbal WM capacity, but cannot speak to the nature of a more domain-general WM as endorsed by other researchers (e.g., Engle, 2002) due to the linguistic tasks utilized.

To circumvent the concurrent and likely inseparable WM and language demands in span tasks, other researchers have employed variants of different WM measures for individuals with aphasia. Gutbrod et al. (1989) presented 60 patients with aphasia, and 36 patients with right-hemisphere lesions, a modified self-ordered pointing task (SOPT, Petrides & Milner, 1982), in which subjects are to point successively to each of a number of pictures presented in a stack of cards without pointing to the same picture twice. The SOPT, a well-known WM task (Baddeley, 2002), requires planning, sequencing, initiating, and monitoring responses, and is sensitive to frontal lobe damage (Spreeen & Strauss, 1998). Although both patient groups performed equally poorly on the most difficult stimuli (photographs of faces), the aphasic group made more errors than the right-hemisphere group on all other stimulus types (line drawings of real objects which could be categorized easily on the basis of either semantic category, visual similarity, or familiar sequences such as letters). Moreover, the aphasic group was less likely to use spontaneously the facilitating strategy of clustering, and made even more errors when experimentally induced to do so. Gutbrod et al. found no apparent differences in performance between patients with Broca's versus Wernicke's aphasia, although patients with global aphasia performed more poorly overall. The aphasic group's deficient performance could not be attributed to semantic impairments, as they demonstrated nearly perfect sorting of the same stimuli. Rather, Gutbrod et al. attributed their

results to an “overload of the central executive” (p. 838) due to impaired phonological and articulatory WM subsystems (cf. Baddeley, 1986). More generally, they suggested that an impaired language system prevented forming a successful plan or strategy for task completion (in this case, deliberate memorization via category clustering). By this hypothesis, individuals with aphasia should be impaired in any high-level cognitive task that requires deliberate or strategic planning, but only to the extent that language skills are (covertly) involved. Another popular WM task, the n-back (described below), was utilized by Downey et al. (2003) in simplified form (i.e., stimuli consisted solely of pictures of common fruits: the “fruit back”) to measure WM in 10 adults with aphasia. These authors failed to find significant correlations between fruit-back performance and subjects’ syntactic comprehension due to low statistical power (data from only five subjects were used for the analysis), but their data suggested decreased WM performance with increasing WM load, as well as decreased comprehension accuracy with increasingly complex sentence construction. Although this suggested a relationship between impaired WM and comprehension, the lack of a comparison group in this study seriously limited its interpretability.

Several studies have taken a mixed perspective by characterizing WM deficits in patients with aphasia differently depending upon lesion site. For example, Beeson et al. (1993) noted mnemonic deficits in their subjects with chronic aphasia (seven patients with anterior and seven with posterior lesions), and concluded that these deficits were “not simply a consequence of the language impairment, but reflect concomitant impairment of memory processes” (p. 274). Although Beeson et al.’s anterior and posterior groups did not differ significantly with respect to language

impairment (measured by the Aphasia Quotient on the WAB, and naming performance), they displayed qualitatively different performance on a verbal memory task. Specifically, whereas the posterior group appeared to benefit from retrieval strategies to improve verbal recall (i.e., guided semantic encoding), the anterior group demonstrated little improvement in recall following exposure to such strategies. A detailed examination of lesion locations in the latter group yielded extension of anterior lesions into the dorsolateral prefrontal region (DLPFC) in six of the seven subjects, with the seventh subject displaying subcortical damage immediately deep to this area. Given the purported role of the DLPFC in EF (see below), these neuroanatomical data supported the authors' contention of EF impairments in the anterior group, dissociated from the patients' language skills, which prevented encoding of stimuli into long-term memory. Posterior patients, on the other hand, demonstrated deficient STM or immediate serial recall, consistent with the proposed role of posterior cortical areas (e.g., parietal cortex) in verbal STM. Recast in Baddeley's (1986) model, the anterior patients demonstrated an impairment of the executive component of WM, whereas posterior patients demonstrated an impaired phonological loop. A similar conclusion was supported by Baldo and Dronkers (1999), who measured verbal and nonverbal (pointing) digit span and spatial span in two groups: patients with left frontal lesions and normal language ( $n = 4$ ), and patients with temporoparietal lesions and conduction aphasia ( $n = 5$ ). Their results demonstrated deficient phonological storage in the patients with conduction aphasia, with a large performance decrement between spatial versus digit spans. Frontal patients, on the other hand, evidenced a milder but cross-modal impairment across

digit and spatial spans, which the authors interpreted as demonstrating an impairment of the central executive with respect to allocating attention during memory tasks.

The ramifications of artificially supporting verbal WM processes during language production for individuals with aphasia have been explored by Linebarger and colleagues (e.g., Linebarger et al., 2007) using an AAC device, the *SentenceShaper*<sup>TM</sup>. This device is proposed to support processing limitations by letting the user monitor his or her speech by providing recurrent WM revivification. This approach is based on the *temporal window hypothesis* of agrammatism (Kolk & van Grunsven, 1985), according to which agrammatic speech and comprehension are the result of rapidly decaying linguistic information, slowed retrieval, or both, preventing holding and integrating in WM the necessary sentence elements. Linebarger et al.'s (2007) approach “artificially enlarges the temporal window for language production” (p. 54) by allowing the user with aphasia to pre-program utterances or sentence fragments into the *SentenceShaper*<sup>TM</sup> device, and then to assemble and replay the utterances (represented by icons) to create multi-sentence utterances. Six patients with chronic, mild-to-moderate nonfluent aphasia demonstrated variable levels of improvement in measures of structure, content, and rate in both their “aided” and unaided” narrative productions (with and without the device, respectively). Interestingly, the amount of device usage was not strongly related to outcome; instead, the authors attributed the variability in their results to “individual subject characteristics, such as motivation and untested cognitive abilities” (p. 62). Of note, the improvements that were noted occurred *in the absence of direct language treatment or cueing*, providing compelling support for the idea of

general performance limitations—versus loss of specific linguistic structures—in generating or exacerbating the symptoms of aphasia (McNeil et al., 1991), and for the specific idea of WM limitations in aphasia. Linebarger et al., however, did not specify whether they conceived of these limitations as specific to the linguistic domain or more generally localized across cognitive domains, nor did they thoroughly test nonverbal cognitive processes in their study participants.

Despite attempts by some of the studies in this literature to avoid verbal WM span tasks, that *all* the studies of WM in aphasia to date have included nameable stimuli (Downey et al., 2004; Gutbrod et al., 1989), verbal response requirements (Beeson et al., 1993), or both (Caspari et al., 1998; Linebarger et al., 2007; Tompkins et al., 1994) makes it difficult to rule out that their results were heavily influenced by linguistic variables inherent in overt or covert verbal encoding (Nystrom et al., 2000). With the exception of Caplan and Waters (1995; 1999), and Gutbrod et al. (1989), who emphasized domain-specific WM systems, the domain-general WM deficits in adults with aphasia reported by these studies may simply have been an artifact or manifestation of primary, linguistic deficits. That is, during a given cognitive task, adults with aphasia may need more resources to perform required or embedded linguistic operations (Just & Carpenter, 1992), leaving fewer resources for other, nonlinguistic processes. Such performance, then, may be interpreted dually as representing either a domain-specific or general WM deficit.

In sum, while the results of most studies suggest a WM capacity deficit may contribute to the language processing difficulties of adults with aphasia, the definition of WM capacity has varied considerably amongst studies (Feldman-Barrett, Tugade,



& Engle, 2004), with authors focusing differentially on either the general “central executive” (Baddeley, 1986; Baldo & Dronkers, 1999; Beeson et al., 1993), aspects of the central executive (Downey et al., 2003; Shallice, 1982), the phonological loop (Gutbrod et al., 1989), or the capacity to recall strictly verbal information (Caplan & Waters, 1999; Caspari et al., 1998; Linebarger et al., 2007; Tompkins et al., 1994; Wright et al., 2003). Moreover, previous research does not easily allow differentiation between this linguistically-mediated WM deficit and a more domain-general loss of WM capacity, because researchers have yet to vary systematically the complexity of linguistic tasks or stimuli, or include stimuli which minimize verbal encoding. This underspecification of the proposed underlying WM deficit in aphasia considerably weakens its power as an explanatory factor in aphasia symptomology.

## 2. The construct of working memory

The conundrum which emerges from the aphasia literature likely stems at least in part from the complexity of the WM construct, which itself is subject to debate in psychological circles. Kimberg, D’Esposito, and Farah (1997) claimed that “if you ask 100 cognitive psychologists what working memory is, you will get 100 different answers” (p. 188). Accordingly, although the term “working memory” is extremely common in the literature, its precise definition varies across different fields of study. Thus, a review of four prominent characterizations of this construct will be presented, followed by an overview of the neuroanatomical basis of WM, and finally, behavioral measures commonly utilized to estimate WM capacity.

## ***2.1 The multi-component model: “Working memory” is storage plus domain-specific processing***

The predominant view of WM was first proposed by Baddeley and Hitch (1974), who questioned the notion of simple STM as an important component of processing systems, given that STM measures historically failed to correlate with higher-level cognitive tasks. As noted by Engle and Kane (2004) in their review of Baddeley’s work, “It is quite unlikely that immediate memory evolved for the purpose of allowing an organism to store or rehearse information (such as a phone number) while doing nothing else. Instead, an adaptive immediate-memory system would allow the organism to keep task-relevant information active and accessible during the execution of complex cognitive and behavioral tasks. The ‘work’ of immediate memory is to serve an organism’s goals for action” (pp. 146-7). Likewise, Baddeley and Hitch explored the idea of an interplay between information storage and processing, and proposed a common WM system “that is limited in capacity and operates across a range of tasks involving different processing codes and different input modalities” (Baddeley, 1986, p.35). The multi-component model of WM consisted originally of a central executive responsible for control processes such as planning or decision-making, and two “slave” systems—a phonological loop and visuospatial sketchpad for temporary storage and maintenance of information. The model follows from a hierarchical view of intellectual function, in which one area of the brain mediates predominately some general, superordinate ability, whereas other regions govern more specific subordinate abilities (Basso et al., 1973).

### *Baddeley's central executive*

Baddeley's (1986) central executive was based on the *Supervisory Attentional System* (SAS, Glosser & Goodglass, 1990; Shallice, 1982; Shallice & Burgess, 1991). Shallice and Burgess (1991) further elaborated this system, proposing that the SAS controlled the following: (1) goal articulation, (2) provisional plan formation, (3) marker creation (i.e., a "message that some future behavior could or even should...be viewed as especially relevant for action" (p. 737)), and (4) marker triggering, by a mental or physical event. In summary, the SAS involves conscious effort and is engaged in novel situations requiring mental flexibility to assemble problem-solving sequences as needed (Kiss, Pazderka-Robinson, & Floden, 2001).

### *Baddeley's recent conceptualization of working memory*

Baddeley and colleagues (Baddeley, 1986, 2000, 2002; Baddeley, Chincotta, & Adlam, 2001; Baddeley, Gathercole, & Papagno, 1998; Baddeley & Hitch, 1994) have further developed the multi-component model, which currently emphasizes structure over function (Baddeley, 2002; Baddeley & Hitch, 1994; Engle & Kane, 2004) and is often associated with a neuropsychological approach (Baddeley & Hitch, 1994). Similar to the original model (Baddeley & Hitch, 1974), more recent characterizations of WM consist of a hierarchical structure with a number of temporary storage systems (i.e., the phonological loop, visuo-spatial sketchpad, and a multi-modal "episodic buffer"), working under a central executive component. The primary role of the phonological loop is now contended to center around acquisition of novel phonological forms, rather than retaining sequences of familiar words

(Baddeley et al., 1998), although it may also play a more central role in coordinating actions during dual tasks (Baddeley et al., 2001). Other researchers have emphasized that the concept of the phonological loop must be tempered by the fact that other forms of language representation (e.g., semantic and lexical factors) have been shown to mediate STM (Collette et al., 2001; Freedman & Martin, 2001; R. C. Martin, Lesch et al., 1999). Baddeley has countered these observations by creating the concept of an “episodic buffer” which allows for multi-modal representations and is capable of integrating information across representational systems and utilizing it across longer time scales than those assumed originally for slave systems or short-term stores (Baddeley, 2000, 2002). Despite his acknowledgement of cross-modal integration, Baddeley has continued to emphasize that these buffers are fractionated from one another and from the central executive (Repovs & Baddeley, 2006). Some recent work by Baddeley and colleagues has suggested fractionation of the central executive itself, into, for example, (1) focused attention capabilities, and (2) dual task management (Baddeley, 2000; Logie, Della Sala, Cocchini, & Baddeley, 2004); other studies, however, have given evidence for a unified executive (Baddeley et al., 2001; Engle, 2002). The central executive, the least specified component of Baddeley and Hitch’s (1974) core WM model, has gradually been defined more specifically over time and is arguably conceptualized currently as purely attentional in nature (Baddeley, 2002; Baddeley et al., 2001).

## ***2.2 The resource-sharing view: “Working Memory” is a unitary system***

Following the logic of Baddeley and Hitch (1974), the core of many WM models is a moment-to-moment trade-off between resources allocated for processing versus storage. Unlike the multi-component model, however, resource-sharing views of working memory reject the notion of “maintaining a clear conceptual and operational distinction between the proposed [WM] subsystems” (Repovs & Baddeley, 2006, p.16), postulating instead a single, unitary system devoid of extraneous storage buffers.

Proponents of these theories view the central executive as a limited capacity space utilized for both storage (e.g., rehearsal strategies) and processing (Just & Carpenter, 1992). An individual is thought to draw on a limited pool of resources to process incoming information, and then to store temporarily partial or final processing products during sequential computations until task completion (Caspari et al., 1998; Just & Carpenter, 1992). Thus, as more effort is required to process information in a given task, less storage space is concurrently available. By defining WM as occurring within a single cognitive workspace, this view necessarily predicts that WM is domain-general in nature, constrained by individual differences in processing efficiency. These individual differences, then, are task-specific: that is, the overall capacity of the central executive does not differ across individuals; rather, differences in WM storage are a function of how much attention (or effort) a particular task requires, depending upon the *domain-specific* abilities of a particular individual (Engle, Nations, & Cantor, 1990).

The resource-sharing view was reflected in the work of Daneman and Carpenter (1980; 1983), which examined the correlation between WM capacity and

reading comprehension in healthy adults. Daneman and Carpenter (1980) designed a now widely used task to measure WM capacity, the reading span task, which required reading aloud sets of sentences and, at the end of a set (varying from three to seven sentences), recalling the last word of each sentence. Critically, the task requires simultaneous processing and storage of information, whereby WM capacity is operationally defined by the number of sentence-final words recalled. Daneman and Carpenter hypothesized that normal variation in reading skill or efficiency in healthy adults would drive the relationship between WM span and reading comprehension, such that adults with higher-level reading skills would be able to read the sentences more efficiently and thus have more storage space available than would adults with less efficient reading processes. Specifically, good readers should recall more sentence-final words than poor readers, because they expend fewer processing resources during the reading task and can devote more WM resources to remembering the words. As predicted, WM span correlated significantly with reading ability, as measured by the verbal portion of the Scholastic Aptitude Test (SAT).

Building upon these data, Just and Carpenter (1992) presented a WM-inspired capacity theory of language comprehension. They suggested that both storage and processing were fueled by a single source of activation and defined capacity as “the maximum amount of activation available in working memory to support either of the two functions” (p. 123). According to these authors, a trade-off between storage and processing occurs when an activation maximum is about to be exceeded, affecting either function approximately equally. They conceptualized individual differences in WM capacity as differences in underlying resource supply, “as though it were an

energy source that some people have more of than other people have” (p. 124). Just and Carpenter discussed an underlying resource supply in general terms, but specified that their WM model was specific to language; in fact, they defined the Reading Span as measuring specifically WM for language versus a more “general” factor.

A number of researchers have found evidence consistent with the resource-sharing account of WM. Hartley and colleagues (Hartley, Speer, Jonides, Reuter-Lorenz, & Smith, 2001), for example, found that processing efficiency, defined as simple reaction time, accounted for over 94% of age-related variance in WM across several different processing codes (e.g., spatial vs. verbal).

### ***2.3 General capacity approach: “Working memory” is executive attention***

A separate approach to conceptualizing WM follows from the assumption that, as a necessary construct involved in everyday activities (Baddeley & Hitch, 1974), WM should be related strongly to broad concepts such as reasoning or general fluid intelligence (Engle, Kane, & Tuholski, 1999; Jarrold & Towse, 2006). As such, many researchers (e.g., Baddeley, 1986; Engle, 2002) suggest that WM is more than the sum of its parts, and, in addition to depending conjointly on processing efficiency (Daneman & Carpenter, 1980; 1983) and storage capacity (Bayliss, Jarrold, Gunn, & Baddeley, 2003), involves critical executive processes necessary for coordinating and integrating storage and processing aspects of a given task. Whereas this view is not wholly incompatible with the original multi-component model of WM, the general capacity approach is not concerned with the structure of individual WM components (e.g., Repovs & Baddeley, 2006), or a trade-off between storage and processing (e.g.,

Daneman & Carpenter, 1980, 1983; Just & Carpenter, 1992), but with what dictates overall capacity and drives the relationship between tasks measuring WM and those reflecting higher cognitive abilities.

Engle and his colleagues, for example, have conceptualized WM as a single, global cognitive capacity which is isomorphic with the construct of executive attention (Cowan, 1998). According to this “general capacity hypothesis” (Conway & Engle, 1996; Hambrick & Engle, 2003), “WM capacity is not directly about memory—it is about using attention to maintain or suppress information.... Greater WM capacity does mean that more items can be maintained as active, but this is a result of greater ability to control attention, not a larger memory store” (Engle, 2002, p. 20). Individuals who perform better on WM tasks simply have more attentional control, allowing them to maintain information in an active, accessible state, and to resist interference, independent of task content. That is, although Engle and colleagues acknowledge that individuals will vary in the efficiency of their mental operations (e.g., reading skill), this normal variation should not affect the correlation between WM capacity and higher-level cognitive task performance. By this line of logic, individual differences in WM capacity reflect differences in executive control to maintain goal-relevant information (i.e., memory representations) in an active or highly accessible state, especially under conditions of competition or interference (Engle & Kane, 2004; Engle et al., 1999; Kane & Engle, 2002). These researchers further maintained that the neurological basis of WM differences is mediated by normal individual heterogeneity in the structure and function of DLPFC (Engle et al., 1999; Kane & Engle, 2002), known to house executive-attention processes. Other



researchers have placed the locus of heterogeneity of function in the anterior cingulate cortex (ACC, Osaka et al., 2003)).

The specific features of this WM model include the following: (1) domain-free, limited capacity controlled attention; (2) long-term memory traces activated above threshold; and (3) domain-specific procedures and skills necessary to achieve and maintain this activation (Engle et al., 1999). Note that the notion of STM is obviated by this model; that is, rather than positing separable STM and LTM stores, these authors followed in the tradition of connectionist models (e.g., MacDonald & Christiansen, 2002), in which STM is conceptualized as that information from LTM which is active above some critical threshold (Feldman-Barrett et al., 2004). By this view, representations can be maintained in many formats, but not necessarily in distinct storage modules (Hambrick & Engle, 2003). The authors stressed further that any domain-specific differences among individuals are much less important for driving WM capacity than abiding, general differences in capacity for controlled processing, and have argued that previous studies demonstrating domain-specific WM effects used subjects with a restricted range of ability ( e.g., undergraduates at a prestigious university; Shah & Miyake, 1996), with the consequence of manufacturing domain-specific effects due to a lack of performance variation (Kane & Engle, 2002).

In a series of studies by Kane, Engle and colleagues, the data supporting their view generally fall along three lines of evidence. First, they have shown that people perform consistently across WM tasks despite different processing demands. For example, Turner and Engle (1989) found that performance on an operation span task,

in which the processing component of Daneman and Carpenter's (1980) reading span task was changed from reading sentences to performing mathematical operations (while concurrently recalling words shown at the end of each mathematical problem), correlated just as well with reading comprehension as did the original reading span task. Importantly, Turner and Engle's results have been supported by others outside of their research group; for example, Waters and Caplan (2003) found that six WM tasks varying in task demands (e.g., subtraction, grammatical judgment) and nature of stimuli (e.g., numbers, words), loaded consistently onto one, general WM factor across subjects of varying ages and abilities (although see Shah and Miyake (1996), who argued that mathematical operations are inherently language-based). Second, several studies have failed to find effects of processing speed or accuracy (i.e., "expertise" in a given processing task) on WM span scores (Conway & Engle, 1996; Engle, Cantor, & Carullo, 1992). Conway and Engle (1996), for example, equated subjects for processing efficiency in the operation span task by adjusting task difficulty (i.e., to obtain 75% mathematical accuracy) across subjects. They found that the storage component of the span tasks (i.e., the number of words recalled) was independent of the level of difficulty of a particular operation, and likewise predicted reading comprehension regardless of processing demand (see also Roberts & Gibson, 2002). Therefore, they argued that it is not the demand of the processing component that is critical for determining WM capacity, but rather the extent to which the processing component requires attention switching or controlled, effortful processing for successful task completion. Finally, systematic differences across individuals with high versus low WM capacity on measures of controlled attention have been

demonstrated. For example, WM capacity as measured by a classic reading span task has been shown to predict normal subjects' performance on subsequent "molecular" attention-demanding tasks, such as anti-saccade and dichotic listening tasks (Kane, Bleckley, Conway, & Engle, 2001; Mitchell, Macrae, & Gillchrist, 2002).

The emphasis on functional and conceptual links between WM and attention was highlighted by Awh, Vogel, and Oh (2006) in their review of attention as a "gatekeeper" for WM. They suggested that attention served two broad purposes during WM tasks: first, to exhibit top-down control over which items will occupy the limited WM space, and second, to bias the encoding of information towards that which is most relevant for current processing goals: in other words, "that which is remembered is also attended" (p. 204). Emphasizing the overlap between neural substrates shown to mediate aspects of WM and selective attention, the authors noted further that "it may not be productive to question *whether* attention plays a role in [WM] executive processes, since it is not clear that there are viable alternatives to this broad proposal" (p. 207).

#### ***2.4 The emergent view: "Working memory" is tied to domain-specific representations***

The emergent view of WM has evolved as a product of the growing number of researchers who have speculated that separate resource pools might be dedicated to different aspects of WM. Shah and Miyake (1996), for example, found that verbal and spatial WM resources were separable in a large sample of college students. They developed a spatial span task, analogous to Daneman and Carpenter's (1980) reading

span, and demonstrated that although spatial span performance correlated with other measures of spatial ability, it did not predict verbal ability; likewise, reading span scores correlated only with verbal, but not spatial, ability measures. These results have been replicated in neuroimaging studies as well (Smith, Jonides, Marshuetz, & Koeppel, 1998).

At a more detailed linguistic level, analyses of sentence comprehension processes have been used to argue for a distinction between WM utilized during syntactic comprehension versus more generic interpretation of input, such as integrating meaning within and across sentences (Caplan & Waters, 1999), as noted above. These and similar analyses have let some researchers to postulate that WM for syntactic analysis should be conceptualized separately from the umbrella of general WM capacity (Jarrold & Towse, 2006).

Crosson et al. (1999), using fMRI to measure WM performance in 12 NBD subjects, further fractionated verbal WM into separate resource pools for semantic, phonological, and orthographic processes. Martin (1995) also contended that separable pools of WM resources mediate different language processes, and that normal individual differences on WM tasks are due to variations in experience or innate factors “such as the richness of neural connections in language areas” (p. 626). Martin and colleagues (Freedman & Martin, 2001; R. C. Martin, 1995; R. C. Martin, Breedin et al., 1999; R. C. Martin & Freedman, 2001) have published a series of studies endorsing this view that verbal WM is inextricably tied to and exists to sustain linguistic representations and processes.

MacDonald and Christiansen (2002) similarly argued that “processing capacity” is not a primitive in and of itself, but rather an emergent property of a system, dependent upon individual biological differences (e.g., differences in the precision of phonological representations) and variations in experience (e.g., exposure to language via reading). They maintained that linguistic knowledge and WM are inseparable, consistent with the close ties between knowledge and capacity in the connectionist approach to language processing. In connectionist models, any “damage” to or manipulations of the system affects the nature of the representations embedded within the network as well as its processing; neither construct is independent from the other. Capacity does not enable certain skills, rather it is synonymous with those skills. Thus the WM construct purportedly measured by tasks such as the reading span reflects simply the specific processing demands of the task itself: “We do not claim that there is a unitary construct called *working memory capacity* measured by working memory tasks any more than we claim that lexical decision tasks measure *lexical decision capacity* separate from language comprehension abilities. Reading span, lexical decision, and reading are all just language processing tasks” (p. 39, italics in original). For example, “high span” individuals (according to performance on a reading span task) who exhibit better performance than low span individuals in understanding object relative constructions do so simply because they likely read more often and thus have more experience with all types of relative constructions (Caplan & Waters, 1995; Friedmann & Gvion, 2003). A similar viewpoint has been advanced by Mainela-Arnold and Evans (2004; 2005) regarding verbal WM or processing limitations in children with Specific

Language Impairment (SLI). These researchers found that previous claims of decreased WM capacity in this population could be explained by differences in long-term lexical knowledge between children with SLI and typically-developing children, with the former group demonstrating significantly poorer recall of low-frequency words compared to their peers (see also Nation et al., 1999).

In the non-verbal domain, Goldman-Rakic and colleagues (e.g., Goldman-Rakic, 1987; Goldman-Rakic, 1993), reviewed by Courtney (2004), defined WM as “sustained active representation of a limited amount of currently relevant information so that it is available for use” (Courtney, 2004, p. 501). Goldman-Rakic and colleagues challenged that super-ordinate executive processes can direct, homunculus-like, the actions of subordinate maintenance systems (Baddeley & Hitch, 1974; Kane & Engle, 2002). Instead, they subsumed concepts such as an individual’s goals, or current contextual demands, under the umbrella of “types of information,” on par with more traditional stimulus-specific features including visual, verbal, or spatial information, to be maintained in WM during a given task. According to this *representational view* of cognition, the type of information received and transformed in each cortical area is the fundamental organizing principle of the brain, rather than the type of processing. Processes such as attention and cognitive control, rather than being contained in any one or particular set of brain regions, are emergent, arising as a consequence of the information sustained in WM at a given moment in time. Although the representational model of WM obviates the need for a separable, executive component in WM, it is otherwise not a radical departure from the neuropsychological concepts of WM reviewed above. In fact, the original SAS

concept (Shallice, 1982) involved delays between a stimulus or memory, and action; likewise, Courtney (2004) noted the ability of PFC to “demonstrate sustained activation representing task-relevant information in the absence of sensory input, or even in the face of distracting, irrelevant sensory input.... This sustained activity is thought to underlie the PFC’s ability to bridge temporal gaps between stimuli and behavior” (p. 501). However, that this view emphasizes WM as a distributed system, emergent from existing levels of processing, distinguishes it from the common, limited capacity system of the multi-component model (Mottaghy, 2006).

That WM performance might simply reflect domain-specific processes has led several researchers to question the utility of a WM construct separate from domain-specific processing capabilities. Postle (2006), for example, summed up the situation by noting that the standard multi-component model of WM has become “a victim of its own success” (p. 23), with difficulty accommodating the findings reviewed above, and other domain-specific fractionations, from the large number of studies that it has motivated. That is, Baddeley and colleagues’ initial approach of deciphering the model’s structure by demonstrating behavioral and neurobiological dissociations (reviewed by Repovs & Baddeley, 2006) seems to require an increasingly complex taxonomy of working memory subsystems linked to the Central Executive. As such, this “subverts a model that once made a strong intuitive appeal to parsimony into an unwieldy organizational scheme that redundantly duplicates every representational system in the mind and brain” (Postle, 2006, p. 25). Therefore, Postle advanced an emergent view of WM, by which it is akin to flexible, selective attention, emerging from a nervous system capable of representing multi-faceted information. At the

theoretical level, this view eliminates the need for specialized storage buffers, and at the neurobiological level, specialized regions of the PFC. Similar to Engle and colleagues (e.g., Engle et al., 1999), Postle reviewed evidence that the contributions of PFC to WM performance likely include mediation of interference and distraction, attention and selection, or flexible control (maintenance of behavioral goals or task rules); but noted that “none of [these] control processes... are specific to or specialized for working memory... the control of working memory does not differ qualitatively from the control of any other behavioral or mental function” (p. 33).

### ***2.5 Summary: Current status of working memory models***

In the years since Baddeley and Hitch (1974) first proposed their WM model, many researchers have analyzed the model’s components and the relationship between WM span measures and other measures of higher-level cognition. The four prominent characterizations of WM reviewed above differ primarily in their views of the source of known individual differences in WM capacity. Specifically, no consensus has been reached as to whether variations in WM reflect a central, domain-free executive or domain-specific components (Engle, 2002). Proponents of the domain-specific view posit multiple resources pools from which individuals draw to mediate storage and processing across different domains (Caplan & Waters, 1995; R. C. Martin, 1995; R. C. Martin & Freedman, 2001), or a single resource pool controlled by domain-specific task demands (Daneman & Carpenter, 1980). According to this view, individual differences on WM tasks may be due to differences in any of the processing or storage components inherent in the task itself



(Bayliss et al., 2003; Shah & Miyake, 1996). Those who endorse a domain-general view of WM, on the other hand, maintain that performance on WM span tasks represents a common factor which is of fundamental importance to general higher-level cognition, such as attention or executive ability (Engle, 2002; Engle et al., 1999; Kane & Engle, 2002).

## ***2.6 Neuroanatomy of working memory***

Goldman-Rakic (1987; 1993) was the first to propose an explicit connection between aspects of the multi-component model (Baddeley, 1986) and the sustained delay period activity of individual PFC neurons studied in monkeys (Fuster, 1973). Since Goldman-Rakic's initial proposal, an explosion of research integrating neuroscientific and psychological approaches to WM has taken place (Aboitiz et al., 2006; Postle, 2006). Results from monkey electrophysiology, experimental psychology, neuroimaging, human electrophysiology, and human neuropsychology have collectively led to what Postle (2006) referred to as the "standard model" of WM, which draws explicit connections between PFC areas mediating WM and projections from posterior processing areas. Despite the widespread influence of this WM model, it is important to note that a controversy exists with respect to the PFC's role. That is, whereas many researchers endorse "the intuitively appealing and parsimonious idea that working memory for different domains of information is accomplished by PFC modules that receive direct projections from specific posterior perceptual information processing areas" (Postle, 2006, p. 24), others specify a domain-general central executive in PFC, "directing" (via, e.g., mediation of

interference and distraction; Engle, 2002) the activity of the “slave” systems in posterior cortical areas. Still others have shown that a more complicated organizational scheme might be in order, with executive WM functions shared between anterior and posterior areas (Kiss et al., 2001), or emergent from interactions between specialized information-processing systems (Adcock, Constable, Gore, & Goldman-Rakic, 2000). By and large, however, basic and clinical neuroscience research has supported the theoretical structure implied by the multi-component model, with executive processes relying on distinct neural substrates from those that maintain information in WM (Awh et al., 2006).

There is general agreement that the WM system is neuroanatomically distributed, involving at a minimum the PFC, ACC, hippocampal cortex, and posterior sensory and motor cortices (Engle & Kane, 2004; Fan, Fossella, Sommer, Wu, & Posner, 2003; Kane & Engle, 2002). Within this network, the executive aspect of WM is classically localized to PFC (Duncan, Burgess, & Emslie, 1995; Goldman-Rakic, 1993; Smith et al., 1998). Neuroimaging studies based on the multi-component model have found that item recognition tasks, requiring primarily storage components, show a left-lateralized pattern of activation in the premotor and supplementary motor (SMA) areas dorsal to Broca’s area. Subtraction of this “rehearsal circuit” (Smith & Jonides, 1999) from the proposed executive-attention processes mediating such tasks (Kane & Engle, 2002) reveals bilateral activation of DLPFC (BA 46) anterior to the activation for storage processes, in addition to activation of premotor cortex, SMA, Broca’s area, and the posterior parietal lobe (Smith & Jonides, 1999). DLPFC activation has been proposed to reflect the

processes of updating, maintaining, or manipulating the contents of WM (Engle, 2002; Engle & Kane, 2004; Nystrom et al., 2000; Sylvester et al., 2003), “task management” or attention allocation across dual task performance (Smith & Jonides, 1999), or representation of current task requirements (Courtney, 2004). That PFC delay-period activity in electrophysiological studies appears to show load dependence in a domain-independent manner (Ranganath, 2006) supports the domain-general nature of this area in supporting WM functions. Finally, premotor cortical activation may also be involved in inhibiting automatic motor responses (Sylvester et al., 2003).

Kane and Engle (2002) presented a detailed review of the neurobiological correlates of WM. Derived from a model of executive and PFC functioning proposed by Cohen and colleagues (Cohen et al., 1994; Cohen et al., 1997; Spreen & Strauss, 1998) they conceptualized PFC “control” capabilities as the active maintenance of task demands amid interference. Specifically, PFC is visualized as acting on representations maintained in posterior and hippocampal cortex, and as such, “PFC dynamically maintains and updates goal information to bias processing in networked areas and to retrieve goal-relevant information from those areas as needed” (p. 642). Thus, controlled or executive processing is emergent from interactions amongst PFC and distributed representations in posterior sensory and motor cortices and the hippocampus. Additional neural structures may influence PFC activation including the thalamus (i.e., sensory-based thalamic gating mechanisms), influenced in turn by the amygdalar complex and basal ganglia, as well as the locus coeruleus, which receives inputs from the amygdala and ACC before projecting to PFC. In this manner,

PFC activation may be affected in a bottom-up manner from sensory input or by top-down influences of learning and past experience (Feldman-Barrett et al., 2004).

Given that PFC is rich in dopamine, Kane and Engle (2002) speculated that dopamine circuits between PFC and midbrain areas (e.g., the ventral tegmental area; VTA) may allow the PFC to increase activity in excitatory or inhibitory loops to maintain or block information, respectively, as needed, thereby biasing signals from other brain areas back to itself (cf. Goldberg et al., 2003). Other researchers have likewise implicated dopamine as a signal-to-noise modulator in task-related PFC neurons, especially during preparation of a behaviorally relevant motor action (Gibbs & D'Esposito, 2006).

Callicott et al. (1999) provided a comprehensive examination of the physiological basis of capacity constraints in WM. Building upon the idea that “working memory capacity might arise from or be coincident with a failure to activate one or more key brain regions during a working memory challenge” (p. 20), the authors used fMRI to measure activation in nine NBD subjects during performance of a parametric n-back WM task. They found a capacity-constrained cortical response to WM load (i.e., an inverted u-shaped neurophysiological response from lowest to highest WM load, with decreased cortical signal coincident with a significant performance decrement at the highest load) in bilateral DLPFC. In contrast, regions such as the pericingulate (particularly ACC) showed a “capacity unconstrained” response (i.e., continuously increasing response with increased WM load), consistent with studies implicating this area for increased effort, attention, or compensation for prefrontal limitations (Osaka et al., 2003). Although Callicott et al.

noted that in addition to DLPFC, regions such as parietal cortex, premotor cortex, and thalamus also exhibited the capacity-constrained response pattern, these responses were significantly less stable than in DLPFC. The authors thus proposed that such activation represented signals “downstream” from DLPFC, although they did not rule out the possibility that capacity constraints could be a network-wide phenomenon. The idea of capacity constraints arising in DLPFC, with functional implications elsewhere, is consistent with Kane and Engle’s (2002) proposal of dopamine-mediated recruitment of cortical areas during WM tasks, and with the proposed role of the DLPFC as the central executive of WM (Baddeley, 1986).

The domain-specific storage and processing components of WM have been shown to be closely linked to neural systems specialized for perception and action (Postle, 2006; Ranganath, 2006). For example, simple maintenance of visual representations has been shown to be associated with persistent activation in inferior temporal cortex, an area considered to be the final stage of the ventral visual pathway (Ungerleider & Mishkin, 1982). Likewise, the neural components of subvocal rehearsal and phonological storage are separable, with the former process housed in Broca’s area, premotor cortex, and SMA (Smith & Jonides, 1999; Smith et al., 1998), and the latter housed in the left inferior parietal lobule, consistent with known roles of these areas in language processing.

## ***2.7 Behavioral measures of WM***

The basis of WM span tasks is to tax the executive control of attention by requiring stimulus information to remain accessible across attention shifts to and from

a given processing task (Engle et al., 1999). One of the most widely used such tasks is Daneman and Carpenter's (1980) reading span test, a dual-task paradigm in which concurrent processing and storage are required. Inspired by this task structure, a number of variations are available (e.g., "operation span" of Turner and Engle (1989), "rotation span" of Miyake and Shah (1996)). Although all of these tasks can successfully identify and characterize WM or attention deficits in NBD and certain clinical populations, such paradigms present four major problems when testing individuals with aphasia. First, classic WM tasks (e.g., the reading span) rely heavily on linguistic skill. For example, Tompkins et al. (1994), who administered a modified auditory version of the Daneman and Carpenter (1980) task, found that within the LHD group, several subjects could not complete the task due to severe spoken language difficulties; that the authors eliminated any potential subjects who could not complete certain WAB spoken language subtests with 100% accuracy suggests that these problems arose even though subjects with moderate to severe aphasia were excluded. Second, purportedly nonverbal tasks (e.g., Miyake and Shah's rotation span) impose highly complex instructions that likely tax the language comprehension skills of most patients with aphasia. Third, any dual task paradigm is susceptible to misinterpretation with respect to resource theories of allocation versus, for example, "bottleneck" theories of processing; that is, a dual task decrement may occur due to response competition or the requirement to shift attention rather than capacity constraints or inefficient allocation strategies (Caplan & Waters, 1999; Murray, 1999; Shuster, 2004). Fourth, the test-retest reliability of span tasks has been questioned

(Waters & Caplan, 1996), which is worrisome given the known day-to-day variability in patients with aphasia (Tseng et al., 1993).

In contrast, the *n*-back task is a parametric WM task (Cohen et al., 1997) that minimizes linguistic load (although see Martinkauppi, Rama, Aronen, Korvenoja, & Carlson, 2000; Meegan, Pure-Stephenson, Honsberger, & Topan, 2003), entails fairly simple instructions (Gallagher, 1994; Levin et al., 2002), and yet has emerged as a valid measure for WM capacity (Downey et al., 2003; Kane et al., 2004; Salthouse et al., 2003). The task requires subjects to judge whether a current stimulus matches one which occurred *n* places back in a sequence of stimuli. The *n*-back requires recognition rather than a recall response, which is especially advantageous for assessing individuals with aphasia, given evidence of relatively intact simple recognition of both concrete and abstract stimuli in these patients (Lang, 1989). Furthermore, the *n*-back paradigm can be used equally well with different stimulus types, such as objects, shapes, or spatial locations (Nystrom et al., 2000).

Although no neuroimaging studies examining the performance of individuals with aphasia on the *n*-back exist, the *n*-back task has been widely used in functional neuroimaging literature with NBD subjects to study the neural basis of WM (Smith & Jonides, 1999), specifically the executive component of WM (Meegan et al., 2003). Such studies consistently report activation of frontal and pre-frontal areas (Kane & Engle, 2002), Broca's area (Friedmann & Gvion, 2003), and other cortical areas implicated in the WM network (Callicott et al., 1999). Critically, pre-frontal activation during the *n*-back task intensifies with increasing *n* values (Simmons, 2001), consistent with the view that the task engages the central executive component

of WM (Jarrold & Towse, 2006; Simmons, 2001; Smith & Jonides, 1997). Several other experimental paradigms have provided additional support for the involvement of this executive component in the n-back task. For example, Watter, Geffen, and Geffen (2001) performed an ERP analysis of n-back task demands, based on the fact that the P300 signal amplitude reliably increases as greater processing capacity is required, but *decreases* during the secondary task in dual task paradigms, when the difficulty of the primary task is increased. Watter et al. concluded that the n-back task fit this dual-task profile. That is, the n-back involved high demands on WM, including sequencing, updating and searching memory contents (Perlstein et al., 2004); and a secondary matching task, with executive attention allocation required across tasks depending on stimulus parameters. Goldberg et al. (2003) likewise described the n-back as a multi-component WM task, involving at a minimum: (1) encoding of stimulus features, (2) temporal indexing, (3) updating (i.e., “target selection and de-selection in the face of competing stimuli” (p. 12), and (4) maintaining information. Accordingly, the n-back fulfills Engle et al.’s (2002) WM requirement of attention shifts during maintenance of stimulus information.

### 3. Linguistic complexity

#### ***3.1 Parameters of linguistic complexity***

Cumulative, long-term linguistic knowledge or lexical organization has been defined by a number of parameters shown to influence both word recognition and production, including neighborhood density, phonotactic probability, phonological complexity, semantic typicality, word age-of-acquisition, familiarity, imageability,



concreteness, visual complexity, and word frequency. *Neighborhood density* refers to the number of words in the phonological neighborhood (i.e., the number of similar-sounding words for a given linguistic item; Morrisette & Gierut, 2002). Words from low-density neighborhoods (i.e., with few phonetically similar counterparts) are recognized and produced more quickly and accurately than words from high-density neighborhoods (Morrisette & Gierut, 2002); the influence of this factor is proposed to occur at the lexical level (Vitevitch & Luce, 2005). *Phonotactic probability* refers to the likelihood of certain sound sequences appearing in words in a particular language, and is contended to facilitate sub-lexical processing (Vitevitch & Luce, 2005).

*Phonological complexity* has been defined as the number of phonemes or syllables in a word (i.e., word length, Mueller, Seymour, Kieras, & Meyer, 2003), or syllable structure (Maas, Barlow, Robin, & Shapiro, 2002). *Semantic typicality* refers to the similarity of a given concept to the prototype (i.e., best or most typical example) of its semantic category (Rosch, 1975). Typicality predicts processing speed across a range of lexical tasks, with typicality and reaction time negatively correlated (Rosch, 1975).

*Age of acquisition* scores are typically derived by asking subjects to use a rating scale to estimate the age at which they learned a given lexical item (Morrison, Ellis, & Quinlan, 1992), and can influence the lexical (phonological) level of word production (Morrison et al., 1992; Nickels & Howard, 1995). *Familiarity* (how often a given lexical item is believed to be seen, heard, or used), *imageability* (how easy it is to create a visual or auditory image of the referent), *concreteness* (how accessible to sensory experience a given referent may be), and *visual complexity* (the number of elements making up a pictured referent) are all typically derived via rating scales

(Nickels & Howard, 1995). Finally, *word frequency* is defined as the frequency with which a given lexical item occurs in the language (Mainela-Arnold & Evans, 2005).

Although these variables have been extensively investigated in both children and adults, limiting the scope to individuals' processing of real words indicates that the most potent factor affecting lexical recognition, recall, and production, with respect to both accuracy and speed, is word frequency (Hulme, Roodenrys, & Mercer, 1995; Jusczyk, 1997; Luce & Pisoni, 1998; Luzzatti et al., 2002; Mainela-Arnold & Evans, 2005; Roodenrys, Hulme, Lethbridge, Hinton, & Nimmo, 2002; Vitevitch & Luce, 2005). Furthermore, word frequency influences the performance of normal adults (Engle et al., 1990) and children with SLI (Mainela-Arnold & Evans, 2004, 2005; Mainela-Arnold, Evans, & Coady, 2005) on verbal WM span tasks.

Word frequency is associated with long-term memory, or word knowledge (Engle et al., 1990; Repovs & Baddeley, 2006), specifically affecting the intactness of structural or semantic representations of words (Luzzatti et al., 2002; Morrisette & Gierut, 2002). It may be encoded as part of the underlying word representation or alternatively as an artifact of lexical activation levels during processing (Morrisette & Gierut, 2002), depending on the specific psycholinguistic theory to which one subscribes. Word frequency also is highly inter-correlated with both rated word familiarity and age of acquisition (Morrison et al., 1992; Nickels & Howard, 1995); in fact, Nickels and Howard (1995) suggested that familiarity may simply be a subjective measure of word frequency. With respect to aphasia, high frequency word representations generally survive brain injury better than low frequency

representations; accordingly, frequency is a robust predictor of naming accuracy in adults with aphasia (Nickels & Howard, 1995; Williams, 1983).

A central tenet of Baddeley's WM model is the existence of the phonological loop, a "slave system" which allows retaining verbal information over short time periods (Baddeley, 1986; Baddeley et al., 1998). In fact, subvocal rehearsal has been shown to be a powerful and highly practiced means for refreshing the contents of memory (Baddeley, 1986; Baddeley et al., 1998; Baddeley & Hitch, 1974), particularly for older adults, who appear to be at a distinct disadvantage when unable to use this tool (Hartley et al., 2001), and especially when memory for order is required (Postle, 2006). Research indicates that covert rehearsal of verbal information in the phonological loop occurs even in the absence of overt demands for spoken word production (Friston et al., 1996; Postle, 2006; Postle, D'Esposito, & Corkin, 2005), and that articulatory coding plays a role in higher level tasks as well (Baddeley et al., 2001; L. B. LaPointe & Engle, 1990). Given the robust effects of word frequency in a wide variety of verbal tasks, similar effects should occur for more complex WM tasks involving recall of verbal information. In fact, Engle et al. (1990) found that word frequency was a robust predictor of performance on a modified version of the verbal span task (although this study also demonstrated a role of an unspecified factor, "working memory capacity," as reviewed in the general capacity model of WM above). Because the n-back task requires serial rehearsal (i.e., continuous updating) of stimuli, it is likely that words of memorized sequences are coded and stored as temporary, phonological representations, and that these memory traces are refreshed by strategic articulatory rehearsal (Goldberg et al., 2003). In sum,

word frequency appears to be a strong correlate of lexical organization as related to WM.

### ***3.2 Non-linguistic stimuli***

The difficulty of “removing” language from any cognitive task is well known (Luria, 1966, 1973; Postle, 2006; Postle et al., 2005). A common practice is utilizing abstract, “non-nameable” shapes (Attneave & Arnoult, 1956); however, researchers have shown that short-term retention of abstract shapes is nevertheless sensitive to verbal distraction (Postle et al., 2005), and may encourage verbal strategies during tasks engendering relatively high cognitive demands, such as the n-back task (Nystrom et al., 2000). This likely reflects the unavoidably high levels of similarity between individual stimuli; that is, internal verbalization may serve to stabilize perceptive discrimination between similar and easily confused patterns (Luria, 1961; 1966).

Given the known problems with using abstract shapes as non-linguistic stimuli, we chose instead to use unfamiliar faces. Such stimuli elicit right lateralized cortical activation during memory tasks (Glogau, Ellgring, Elger, & Helmstaedter, 2003; Kelley et al., 1998), suggesting a nonverbal, icon-like representation when delay intervals are brief, as in an n-back task (Haxby et al., 1996). Thus, individuals with left-hemisphere damage might not experience a disadvantage in processing these types of non-linguistic stimuli as compared to other perceptually similar or visually complex shapes.

#### 4. Summary and proposed hypotheses

Given the domain-specific views of WM reviewed above, in which WM capacity is tied to, or synonymous with, the processing demands of a given task, individuals with aphasia should demonstrate clear WM deficits in any task involving linguistic stimuli, whether by overt or covert verbalization. Furthermore, patients with aphasia should be proportionately impaired on verbal WM tasks with respect to aphasia severity, or their ability (or lack thereof) to utilize or process the linguistic stimuli at hand. Stated another way, the processing demands or difficulty of a given task, according to individual differences in domain-specific skills required for the task, should affect WM capacity.

The theories proposed by Engle and colleagues, on the other hand, lead to separate predictions regarding the relationship between aphasia and WM impairment. According to their interpretation of WM capacity as a domain-free mechanism that reflects PFC executive-attention capabilities, patients with aphasia should demonstrate WM deficits only to the extent that they show concomitant PFC damage or attentional dysfunction. Their specific language abilities, while affecting the domain-specific components of a given WM task (e.g., the ability to read sentences or say aloud sentence-final words), should not drive the relationship between WM capacity and higher-level cognitive function (see Engle et al., 1990).

In summary, the WM literature over the last three decades fractionates into two general views to account for the close ties between WM and higher-level cognitive tasks: (a) a *domain-specific* view, in which one's skill in a specified domain (e.g., language) should drive WM capacity and other, related cognitive measures

which also presumably require that skill; and (b) a *domain-general* view, in which WM capacity is considered an abiding and domain-free characteristic of the attentional focus and inhibitory skills required for many WM tasks and other higher-level cognitive tasks. This debate is directly analogous to the two perspectives from which WM limitations and associated cognitive problems in aphasia have been viewed: i.e., the *domain-specific* and *general capacity* hypotheses, respectively. Thus, whereas many researchers have examined the relationship between WM and language in aphasia, few have considered the nature of the WM deficit itself: that is, whether a WM impairment might stem from a primary language deficit (e.g., Caplan & Waters, 1999), or whether the deficit is secondary to a general, global deficit in executive attention (Engle, 2002). The question that naturally arises is: which WM perspective, domain-specific or domain-general, can better capture the nature of WM impairments in aphasia? This query has theoretical implications for the nature of WM and the nature of aphasia itself, and clinical implications regarding the way in which such deficits should be approached. Specifically, a language-mediated view of WM allows for such “nonlinguistic” cognitive deficits to be constrained by the nature of the primary language impairment in aphasia, implying that language treatment should engender improved WM function. On the other hand, a domain-general view of WM suggests that aphasic impairments are the product of, or exacerbated by, global deficits in nonlinguistic cognition (e.g., McNeil et al., 1991), which may require treatment separately or in addition to treatment for language deficits.

The debate turns on to the extent to which WM deficits experienced by many individuals with aphasia are specific to the verbal domain. To address this matter, we

tested WM capacity across adults with left-hemisphere damage and aphasia (LHD), and control subjects without brain damage (NBD), while manipulating systematically WM load and linguistic stimulus complexity. WM load was manipulated in an n-back task via parametric variation of  $n$  (described below). Linguistic complexity was addressed by manipulating frequency of occurrence using the Kucera-Francis (1967) measure of written word frequency, with words matched on a number of other lexical factors (e.g., length, concreteness, age of acquisition, neighborhood density, and phonotactic probability). Effects of linguistic complexity manipulations were tested for each subject via a confrontation naming task, using the same stimuli designed for the n-back task. Finally, unfamiliar faces were included as non-linguistic stimuli.

The following hypotheses were formulated according to the two competing theoretical accounts, domain-specificity and domain-general, of WM in aphasia. Given the known WM problems in aphasia, adults with left-hemisphere damage and aphasia (LHD) were expected to perform more poorly than healthy, non-brain-damaged adults (NBD) across WM tasks. What varies across the following scenarios is the nature of this depressed performance.

- (i) *Domain-specific view*: LHD individuals demonstrate higher-level WM problems to the extent that they must use their impaired linguistic system (either overtly or covertly) to complete WM tasks.
  - a. A significant interaction will occur between group and language complexity, such that the LHD group will be affected more than the NBD group by increasing linguistic stimulus complexity of (high- to low-frequency) during n-back tasks

- b. The LHD group will not be significantly more impaired on n-back tasks using the nonlinguistic face stimuli compared to the linguistic stimuli.
  - c. In the LHD group, aphasia severity will be more strongly correlated with WM performance than measures of non-verbal problem solving (e.g., *Raven's Colored Progressive Matrices*, RCPM).
- (ii) *Domain-general view*: LHD individuals demonstrate higher-level WM problems that reflect a general decrease in WM capacity
  - a. A significant interaction between group and WM load will occur, such that the LHD group will be more affected by WM load (i.e., LHD performance decrements will be steeper than NBD decrements with increasing values of  $n$ ).
  - b. LHD and NBD groups will demonstrate parallel patterns of performance across levels of linguistic complexity during the n-back tasks (i.e., no significant interaction between language complexity level and group).
  - c. The LHD group will be significantly impaired on nonlinguistic as well as linguistic n-back tasks.
  - d. Measures of non-verbal problem solving, but not aphasia severity, will correlate significantly with WM performance in the LHD group.
- (iii) *Mixed support*: LHD individuals are affected by a combination of linguistically-mediated WM problems in the context of a generally decreased WM capacity.



- a. Significant interactions will occur between (1) group and language complexity level, and (2) group and WM load.
- b. Both aphasia severity and non-verbal problem solving will correlate with WM performance.

## **II. Methods**

### **1. Participants**

Fifteen patients diagnosed with mild to moderate aphasia of various types and twelve healthy, age- and education- matched control subjects participated. One patient failed to complete a portion of the study protocol due to scheduling conflicts, so the data of that individual were excluded from most analyses, reducing the patients' sample size to 14. All subjects with aphasia were recruited from two Midwest cities: Bloomington, Indiana; and Madison, Wisconsin. Control subjects were recruited from these two cities and additionally from Chicago, Illinois.

All participants in the aphasia group suffered a unilateral, left-hemisphere lesion, were at least three months post-onset, and were medically stable. Exclusion criteria included a medical diagnosis of comorbid cognitive deficits (e.g., dementia), or receptive language difficulties so severe as to preclude understanding of informed consent or task instructions. No participants in either the aphasia or control group had a history of traumatic brain injury or other diagnosable neurological or psychiatric conditions. Finally, all subjects completed informal visual and hearing screens, consisting of simple picture matching (Murray, unpublished) and the Speech Discrimination Subtest of the *Arizona Battery for Communication Disorders of*

*Dementia* (ABCD, Bayles & Tomoeda, 1993), respectively, to ensure adequate peripheral skills. The demographic and clinical characteristics of the two samples are presented in Tables 1 and 2.

All study procedures were approved by the Institutional Review Boards at Indiana University (Study # 05-9791) and the University of Wisconsin-Madison (Study SE-2005-0179). Each participant signed an informed consent form before the experiment (Appendix A) and received nominal financial compensation for taking part.

## 2. Tasks

### *2.1 Standard Tests of Aphasia and Cognition*

Subjects with aphasia completed the *Western Aphasia Battery* (WAB, Kertesz, 1982) to characterize the nature and severity of their language deficits. All subjects completed either the *Raven's Colored Progressive Matrices* (RCPM, J. Raven et al., 1998) or the *Test of Nonverbal Intelligence- 3* (TONI-3, Brown, Sherbenou, & Johnsen, 1997) to provide a global measure of non-verbal problem solving and higher-level cognition.

### *2.2 Experimental tasks*

All participants completed a confrontation-naming task for the linguistic stimuli (48 pictured objects, described below) used subsequently in the n-back tasks. The pictures were displayed individually on an i-book G4 laptop computer. Participants were not given a formal time limit for naming; however, following the

procedures commonly used with aphasia naming tests (Kaplan, Goodglass, & Weintraub, 1983), participants who could not name an item within 10 seconds were given a standardized, livevoice semantic cue, then an initial phonemic cue, initial grapheme (written) cue, and the opportunity for repetition of the target name.

All participants also completed a set of three n-back tasks, described below, as a measure of WM performance. N-back tasks included the following stimuli: (1) high-frequency, nameable objects, (2) low-frequency, nameable objects, and (3) non-nameable faces. Parametric variation of WM load (0, 1-, and 2-back) was fully crossed with stimulus type, yielding nine separate n-back tasks per subject.

### *2.2.1 Stimuli*

#### *2.2.1.1. Linguistic Stimuli*

Although it is common to utilize printed words or letters in linguistic n-back tasks (Roberts & Gibson, 2002; Smith & Jonides, 1999), we used photographs of nameable objects to minimize confounds given the frequent co-occurrence of alexia with aphasia. That subjects tend to phonologically recode visually presented, nameable shapes or objects (Baddeley, 2000; Hartley et al., 2001; Postle, 2006; Postle et al., 2005; Repovs & Baddeley, 2006) allowed the pictured items to be inherently “linguistic” without confounding semantic or phonological representations with orthographic decoding skills.

A pool of 45 linguistic stimuli were selected from a larger corpus of words gathered for a separate study (Evans, Coady, Sizemore, & Mainela-Arnold, in prep). These stimuli consisted of regular, one-syllable words, and were balanced with

respect to frequency, familiarity, neighborhood density, phonotactic probability, concreteness, age-of-acquisition, letter length, and number of phonemes (Coltheart, 1981; Kucera & Francis, 1967; Nusbaum, Pisoni, & Davis, 1984).

Written word frequency was chosen as the manipulated linguistic parameter. Accordingly, the linguistic stimuli were divided into two groups according to Kucera-Francis (1967) written word frequency counts; low and high-frequency words were operationally defined as counts  $\leq 20$  and  $\geq 70$ , respectively, following criteria identified in the literature (Kay, Lesser, & Coltheart, 1999). To ensure equally-sized stimulus groups, three more high-frequency stimuli were selected from a separate corpus of balanced words designed for cognitive-neuropsychological assessment of aphasia (Kay et al., 1999).

The selected words were represented by photographs downloaded from the Internet. All photographs were converted to gray scale, with a plain white background, and edited using Microsoft Picture-It! Photo 7.0, then uploaded into Psyscope Beta II (Bonatti, 2006) and adjusted for image size and position on the computer screen (*port* set to: 512, 575, 384, 550, 0). Pictured stimuli were then piloted with a group of 30 adult volunteers (undergraduate students at the University of Wisconsin-Madison) to check for imageability and naming agreement. Stimuli with less than 90% naming agreement were discarded, modified, or replaced, yielding a total of 48 black-and-white pictured objects (representing 24 high-frequency and 24 low-frequency words). A list of stimulus characteristics is displayed in Table 3, and a complete stimulus list is given in Table 4. Notably, the two groups of stimuli did not differ in age of acquisition,  $t(16) = 1.9, p = .07$ , concreteness,  $t(40) = .65, p = .52$ ,

imagery,  $t(40) = 1.8, p = .09$ , letter length,  $t(46) = .72, p = .07$ , or number of phonemes,  $t(46) = .07, p = .80$ . The groups differed significantly in both frequency,  $t(45) = 4.5, p < .01$ , and familiarity,  $t(42) = 6.0, p < .01$ .

#### *2.2.1.2. Nonlinguistic Stimuli*

Nonlinguistic n-back stimuli consisted of a set of 24 pictures of faces (all with neutral facial expression) selected from a larger set of pictures validated for facial affect (Ekman & Friesen, 1976), and used previously to study WM, facial processing, and emotional development and recognition (Evans, Turkstra, & Pollak, in prep; Pollak & Friesen, 2003). The faces (12 males, 12 females; 16 Caucasians, 8 Asians) had been scanned into Adobe Photoshop 6.0, recreated in gray scale, and edited to remove nameable features surrounding the face (e.g., hair, shoulders) These stimuli were uploaded into Psyscope Beta II (Bonatti, 2006), adjusted for image size, and aligned on the computer screen similarly to the procedures described above.

#### *2.2.2 N-back Task Procedures*

The basis of the n-back task is to judge whether a current stimulus matches one which occurred  $n$  places back in a sequence. As described above, study participants completed nine n-back tasks: three levels of WM load (0, 1, and 2) with three levels of linguistic complexity (high-frequency words, low-frequency words, and faces) per load. In the 0-back condition, the target was any stimulus that matched a pre-specified stimulus (e.g., “dog”). In the 1-back condition, the target was any stimulus that matched the one immediately preceding it (i.e., one trial back). In the 2-

back condition, the target was any stimulus identical to one presented two trials back (see Figure 1).

Prior to the experimental n-back tasks, participants received an in-depth tutorial to ensure adequate understanding of the instructions and task format (see Appendix B for script). Instructions were given using both pictured examples and demonstration to minimize the possible effects of auditory comprehension deficits in the aphasia group. Task instructions emphasized both accuracy and speed. All participants completed three practice trials prior to each n-back triad (0-, 1-, and 2-back), including the following:

- 1) A sample sequence to ensure understanding of the 0-, 1-, or 2-back pattern, using simple shapes as stimuli (i.e., circles, squares, triangles, and hearts). The shapes were pictured on a paper strip which was threaded through a cardboard “screen,” and participants were required to point to target stimuli;
- 2) A second sequence to ensure adequate recognition of individual stimulus items, using samples of face stimuli printed out individually on 8 ½ x 11” paper. Participants pointed to target stimuli while the examiner flipped through the pictures; and
- 3) A ten-item computer trial consisting of the actual stimuli and task format. Participants did not go on to the formal tasks until all three practice sets were completed with 100% accuracy.

The n-back task was presented to subjects using Psyscope Beta II (Bonatti, 2006) on an i-book G4 laptop computer. Each stimulus was displayed for 800 ms, with an inter-stimulus interval of 1600 ms. This relatively rapid presentation rate was

chosen to discourage attempts to covertly verbalize the nonlinguistic face stimuli (note that it was expected that participants would covertly verbalize the nameable stimuli). Participants sat at a comfortable distance from the screen with their unaffected or dominant hand resting on the keyboard and pressed a brightly labeled key (located on the “/” or “z” key for right or left-hand preference, respectively) whenever they saw a target stimulus.

Consistent with the n-back literature (e.g., Cohen et al., 1994; Cohen et al., 1997; Perlstein et al., 2004), hit rate ( $p = .33$ ) across n-back tasks was equated by varying the length of each n-back sequence according to WM load, with 100 trials at  $n = 0$ , 160 at  $n = 1$ , and 250 at  $n = 2$ . There were 24 possible unique matches for each n-back task (i.e., 24 stimuli within each stimulus set). For the 0-back tasks, just one match to a pre-specified target was required, with 21 hits possible. Unique matches were duplicated once to yield 48 hits on the 1-back tasks. For the 2-back tasks, some stimuli used as hits were repeated a total of three times to yield 60 possible hits. Across the n-back tasks, non-target stimuli contributed the same number of times to each condition and were distributed approximately equally across each of the sequences.

The n-back tasks were administered in a partially fixed order to limit within-set confusion. That is, participants completed all three 0-back tasks before completing the 1-back tasks and then the 2-back tasks, respectively. Within each triad, the order of tasks by language complexity (high-frequency stimuli, low-frequency stimuli, and faces) was randomized. A rest break was allowed after each sequence as the participant desired. Most participants completed the 0- and 1-back tasks, aphasia and

cognitive tests, and the experimental picture naming task in one 90 min session and the 2-back tasks, in addition to any unfinished paper tasks, in a second 90 min session scheduled approximately one week after the first session.

### *2.2.3 Scoring Procedures*

#### *2.2.3.1. Picture-Naming task*

Following the procedures on the WAB (Kertesz, 1982), each correctly-named item received a score of 3, for a total possible score of 144 across 48 items. Minor articulatory errors were not penalized, but items named with frank phonemic or semantic paraphasias received a score of 2, and items elicited following a phonemic cue received a score of 1. Items received a score of 0 if named correctly only after repetition, if never named following all cues.

#### *2.2.3.2. N-back tasks*

Three dependent measures were chosen to analyze n-back data: reaction time (RT) per each of the nine n-back conditions and two signal detection statistics, Pr (accuracy) and Br (bias). Both Pr and Br were calculated using two probabilities: Hit rate (i.e., the probability of selecting a target), and False Alarm rate (i.e., the probability of selecting a non-target). The probabilities of hit rate and miss rate summed to 1.0 as did the probabilities of false alarm and correct rejection.

Pr was the probability that an item would cross a recognition threshold and was calculated as follows:

$$\text{Pr} = (\text{Hit rate}) - (\text{False alarm rate}) \quad (1)$$



Pr is comparable to  $d'$ , which uses the z-scores of these same values ( $d' = z(\text{Hit rate}) - z(\text{False alarm rate})$ ). Br measured the bias towards a positive or negative response, calculated as:

$$\text{Br} = (\text{False alarm rate}) / (1 - (\text{Pr})) \quad (2)$$

To adjust for Br calculations, a hit rate equal to 1.0 was readjusted to equal .99, and false alarm rates of .00 were increased to .01. The higher the Br value, the more liberal the response (e.g.,  $\text{Br} = 1$ ), and the lower the Br value, the more conservative the response (e.g.,  $\text{Br} = 0$ ). Taken together, Pr and Br account for performance across all possible conditions (hits, misses, false alarms, and correct rejections), unlike percent correct, which does not take into consideration response bias.

### 3. Data Analyses

The study design was a 2x3x3 mixed factorial, with group (LHD or NBD) as the between-subjects factor and three levels of language (high frequency objects, low frequency objects, and faces) and WM load (0-, 1-, and 2-back) as the within-subject factors. Accuracy (Pr), bias (Br), and latency data (RT) from each group were modeled using separate repeated measures analyses. Additionally, data from each group were subjected to a correlational analysis to explore relationships between experimental tasks, standard test performances, and subject characteristics (e.g., age). Finally, given the exploratory nature of this study, an evaluation of the direction of scores in both groups and calculation of effect sizes were completed.

### III. Results

#### 1. Picture Naming Task

As expected, the NBD group performed at ceiling on the picture naming task ( $M = 144$ ,  $SD = 0$ ), whereas the LHD group performed less accurately ( $M = 115.87$ ,  $SD = 33.72$ ). The LHD mean picture-naming score reflected more accurate naming of high- ( $M = 60.87$ ,  $SD = 17.48$ ) versus low-frequency objects ( $M = 55$ ,  $SD = 17.27$ ), as confirmed by a paired-sample t-test,  $t(14) = 2.71$ ,  $p = .02$ ,  $d = .70$ .

#### 2. N-back Tasks

##### 2. 1 Group effects: *Pr*

A significant effect of group,  $F(1, 24) = 24.9$ ,  $p = .00$ ,  $\eta^2 = .51$ , was observed, with the LHD group performing more poorly than the NBD group across n-back tasks (see Tables 5 and 6). As seen in Figure 2, this effect was qualified by a significant interaction between WM load and group,  $F(2, 48) = 17.09$ ,  $p = .00$ ,  $\eta^2 = .42$  such that a larger difference between groups was obtained at higher levels of WM load.

##### 2. 2 Language effects: *Pr*

As shown in Figures 3 and 4, there were significant effects of language condition,  $F(1.6, 39.1) = 24.93$ ,  $p = .00$ ,  $\eta^2 = .51$  with no interaction between language and group,  $F(1.6, 39.3) = 1.57$ ,  $p = .22$ ,  $\eta^2 = .06$  (degrees of freedom adjusted using the Huynh-Feldt procedure for sphericity violations; Huynh & Feldt, 1976). Post-hoc pairwise comparisons (adjusted for multiple comparisons using the

Bonferroni correction) revealed that the locus of this effect was restricted to the difference between faces versus both sets of nameable objects (mean differences of .095, .091,  $p = .00$ , for faces versus high- and low-frequency stimuli, respectively), with no differences between high- versus low-frequency object stimuli (mean difference = .004,  $p = .7$ ). That is, all subjects performed less accurately for the face (non-nameable) stimuli compared to both sets of object (nameable) stimuli across  $n$ -back conditions. These data were confirmed by a restricted,  $2 \times 2 \times 3$  repeated measures ANOVA using only two levels of language load (high- versus low- frequency objects), in which language load failed to reach significance,  $F(1, 24) = .15$ ,  $p = .7$ ,  $\eta^2 = .01$ .

To determine whether the null effects of word frequency were due to near-ceiling performance in the NBD group (especially at lower levels of WM load,  $n$ ), follow-up analyses were completed separately for the LHD group alone. A restricted model repeated measures ANOVA ( $2 \times 3$ ), using two levels of language load (high- versus low- frequency objects) was applied; again, the effects of high versus low frequency stimuli failed to reach significance,  $F(1, 13) = .32$ ,  $p = .58$ ,  $\eta^2 = .02$ . To rule out floor effects at the 2-back level, this model was re-run as a  $2 \times 2$  repeated measures ANOVA (high- and low-frequency objects at WM load levels  $n = 0, 1$ ); once again, only WM load reached significance,  $F(1, 25) = 8.53$ ,  $p = .01$ ,  $\eta^2 = .25$ , and language effects remained negligible,  $F(1, 25) = .16$ ,  $p = .69$ ,  $\eta^2 = .01$ .

### 2. 3 WM load effects: *Pr*

As observable in Figures 2 and 4, the ANOVA analysis revealed a significant main effect of WM load (adjusted using the Huynh-Feldt procedure),  $F(1.6, 39.1) = 96.79, p = .00, \eta p^2 = .8$ , qualified by significant interactions between language and working memory load,  $F(4, 96) = 8.58, p = .00, \eta p^2 = .26$ , and group and WM load,  $F(1.6, 39.1) = 17.09, p = .00, \eta p^2 = .42$ . Post-hoc, pairwise comparisons (adjusted using the Bonferroni correction) revealed significant differences between all WM load levels: Subjects' performances were affected significantly by moving from the 0- to 1- to 2-back tasks. Similar to the language load results, visual inspection of the data revealed that the source of the language by WM load interaction could be pinpointed almost entirely to the differences between faces versus both sets of object stimuli, with subjects across groups experiencing the steepest performance decrement at the 2-back level of face stimuli (see Table 5). This interaction effect remained significant when analyses were restricted to the LHD group alone,  $F(4, 9) = 3.56, p = .013, \eta p^2 = .23$ , and visual inspection of these data revealed parallel trends to the collapsed group data (see Table 6).

To further qualify these data, post-hoc analyses were completed for the LHD group after dividing the subjects by aphasia type (fluent versus nonfluent;  $n = 7$  per group). This 2x3x3 analysis yielded no significant differences in performance between fluent and nonfluent LHD subjects,  $F(1, 12) = .53, p = .48, \eta p^2 = .04$ , and no significant interactions between aphasia type and language level nor aphasia type and WM load. There was a strong main effect of WM load,  $F(2, 24) = 76.32, p = .00, \eta p^2 = .86$ , and language level,  $F(2, 24) = 13.16, p = .00, \eta p^2 = .52$ ; post-hoc contrasts

localized the latter effect strictly to the difference between nameable objects and faces, with no differences between the high- and low-frequency object conditions, analogous to the full model analyses. These effects were qualified by a significant interaction between WM load and language level,  $F(4, 48) = 3.56, p = .01, \eta^2 = .23$ , such that, as in the full model, subjects experienced a steeper performance decrement with respect to faces versus nameable objects at higher WM load levels ( $n > 0$ ).

#### 2. 4 Reaction time (RT)

ANOVA analyses revealed significant main effects of WM load,  $F(2, 46) = 32.07, p = .00, \eta^2 = .58$ , and language condition,  $F(2, 46) = 21.01, p = .00, \eta^2 = .48$ , and a significant WM load by language condition interaction,  $F(4, 92) = 4.95, p = .00, \eta^2 = .18$ . Analysis of these effects indicated that both groups displayed longer RTs as WM load increased, and displayed slower RTs to face versus object stimuli, with a greater difference between face and object stimuli at lower WM load levels. The main effect of group was not significant,  $F(1, 23) = 2.28, p = .15, \eta^2 = .09$ .; however, visual inspection of the data revealed a trend toward longer LHD group RTs compared to the NBD group across conditions (see Figures 5 and 6).

To explore whether the null effects of word frequency on accuracy (Pr) were reflected in RT in the LHD group alone, this group's data were subjected to a restricted ANOVA with two language condition levels (high vs. low frequency) and three WM load levels as the within-subjects factors. Results of this analysis verified the full model RT results and the Pr data, with no significant RT differences between high- versus low-frequency stimuli,  $F(1, 13) = .79, p = .39, \eta^2 = .06$ . WM load

effects remained significant,  $F(2,26) = 29.09$ ,  $p = .00$ ,  $\eta^2 = .69$ , reflecting a moderate increase in RT from 0- to 1-back tasks and large increase from 1- to 2-back tasks (see Figure 6A).

A correlational analysis between RT and Pr data revealed that these two variables were significantly and negatively correlated only at the 1-back level,  $r = -.52$ ,  $p = .01$ . Correlations for the other WM load levels followed a similar trend, but failed to reach significance,  $r = -.35$ ,  $p = .08$ ;  $r = -.36$ ,  $p = .07$  for the 0- and 2-back tasks, respectively.

## 2. 5 Correlational analyses

Correlational analyses were carried out to explore factors influencing the LHD group's performance on the n-back task. Due to the exploratory nature of the analysis, a strict, 2-tailed significance criterion was adopted, and the Bonferroni correction for multiple comparisons was used (Dunn, 1961). Selected results are displayed in Table 7. N-back accuracy scores (Pr) at the 0- and 1-back levels were highly intercorrelated, but neither correlated significantly with the 2-back task. Likewise, accuracy scores for *Pr high*, *Pr low*, and *Pr face* (Pr summed across 0-, 1-, and 2-back tasks using high-frequency, low-frequency, and face stimuli, respectively) were highly intercorrelated. Nonverbal problem-solving (RCPM) was not significantly related to n-back accuracy but was associated with *RT 2* (RT summed across language conditions at the 2-back level). Not surprisingly, aphasia severity (AQ) was significantly related to performance on the experimental picture-naming task; AQ was also associated with *Pr face*, as was picture-naming performance, and LHD subjects'

age. None of these variables, nor any other performance indices, were related to n-back performance with linguistic stimuli. A second correlation analysis, controlling for potential age effects, continued to yield a significant and slightly stronger relationship between AQ and *Pr face*,  $r = .68$ ,  $p = .01$ . To confirm and extend these results, a correlational analysis was run with NBD group data. As shown in Table 8, this analysis likewise yielded a significant relationship between age and *Pr face*, in addition to age and *Pr 2* (Pr summed across language conditions at the 2-back level). Again, whereas *Pr high*, *Pr low*, and *Pr face* scores were highly intercorrelated, n-back performance indices limited to linguistic stimuli (*Pr high*, *Pr low*) did not correlate significantly with any NBD performance measures.

## 2.6 Br (response bias)

ANOVA analysis of the Br data yielded significant main effects of group,  $F(1,23) = 7.67$ ,  $p = .01$ ,  $\eta^2 = .25$ , and WM load,  $F(2,46) = 40.45$ ,  $p = .00$ ,  $\eta^2 = .64$ , and a significant interaction between WM load and language condition,  $F(4,92) = 4.09$ ,  $p = .00$ ,  $\eta^2 = .15$ . The main effect of language condition did not reach significance,  $F(2,46) = 1.56$ ,  $p = .22$ ,  $\eta^2 = .06$ . The significant interaction effect indicated a greater spread of bias scores during the 1-back task versus 0- and 2-back tasks, with more conservative performance for face stimuli compared to object stimuli. As seen in Figure 7, both groups grew relatively more conservative (i.e., fewer false positives, increased number of missed targets) as WM load increased, with the LHD group performing more conservatively than the NBD group overall. A correlational analysis between Br and Pr yielded significant relationships between

these two variables,  $r = .65, p = .01$ ;  $r = .63, p = .00$ ; and  $r = .47, p = .02$  at the 0-, 1-, and 2-back levels, respectively.

## 2. 7 *Vigilance*

Because the 2-back task was necessarily longer than the 0- and 1-back tasks (see Methods, section 2.2.2), and overall vigilance has been implicated in aphasic attentional deficits (Laures, 2005; Laures et al., 2003), the 2-back task data were divided in half (i.e., the first halves of each of the three 2-back conditions, per subject, were pooled and compared to the second halves) and analyzed post-hoc to explore possible task decrement effects. Across LHD subjects and linguistic conditions, the mean Pr scores were .58 (SD = .03) and .52 (SD = .03), respectively, for the first and second halves of the 2-back tasks. A paired t-test yielded significant differences between these scores,  $t(41) = 2.86, p = .01$ . That is, the LHD group performed significantly worse during the second compared to first half of the 2-back tasks. The NBD group did not demonstrate a similar effect, nor was such an effect visible for the 1-back tasks for either the LHD or NBD groups. When the LHD 2-back data were divided according to language level (high-frequency, low-frequency, and faces), however, the vigilance effect disappeared for all but the low-frequency 2-back,  $t(13) = 2.3, p = .04$ . Therefore, attentional vigilance was not a consistent predictor of performance across language levels in the 2-back tasks compared to the 0- and 1-back tasks.



## 2. 8 Reliability

Approximately 25% of the LHD participants were re-tested a minimum of four weeks following their completion of the study protocol to assess the n-back's test-retest reliability with respect to RT and the two signal detection statistics, Pr and Br. Results demonstrated high reliability for Pr,  $r = .93$ ,  $p = .00$ , and RT,  $r = .91$ ,  $p < .00$ , and moderately high reliability for Br,  $r = .73$ ,  $p = .00$ .

## IV. Discussion

The purpose of this study was to disentangle the domain-specific versus domain-general hypotheses regarding the nature of WM impairments in aphasia, by manipulating parametrically domain-specific (language) and domain-general (WM load) factors in a WM task. Collectively, these results supported a domain-general viewpoint, lining up clearly with the second of the three possible scenarios considered: that is, LHD individuals demonstrated higher-level WM problems consistent with a general decrease in WM capacity. Specifically:

- a.) A significant interaction between group and WM load occurred, such that the LHD group was more affected by WM load (i.e., LHD performance decrements were steeper than NBD decrements with increasing values of  $n$ ).
- b.) LHD and NBD groups demonstrated parallel n-back performance patterns across levels of linguistic complexity (i.e., no significant interaction between language complexity level and group).

c.) The LHD group was significantly impaired, compared to the NBD group, on nonlinguistic as well as linguistic n-back tasks.

Results were mixed with respect to the fourth prediction that measures of non-verbal problem solving, but not aphasia severity, would correlate significantly with WM performance in the LHD group. Indeed, this group's RCPM scores were associated with 2-back RT, but not accuracy. Furthermore, their AQ scores (aphasia severity) correlated solely with accuracy on the non-nameable (face) but not the nameable (object) n-back tasks. A secondary purpose was to examine the reliability of this WM task, the n-back, for adults with aphasia. The study results suggested that the n-back is a reliable indicator of WM in aphasia.

The following section will review the implications of these results with respect to the role of WM in aphasia. The evidence for domain generality of WM will be presented and a broader picture of nonverbal cognitive deficits in aphasia will be explored, followed by a review of the remaining case for domain specificity.

## 1. Evidence for domain-general

### ***1.1 Group and WM load effects***

According to Navon (2004), an operationally defined, attention-dependent disorder should be “manifested mainly in specific conditions conventionally thought to constrain attention (e.g., high load)” (p. 840). This phenomenon is exactly what was observed in the present set of results: Adults with aphasia performed poorly on the n-back task, compared to healthy controls, and more poorly still, comparatively, under conditions of high WM load. The nominal effects of varying linguistic

complexity (word frequency) across n-back tasks demonstrated that WM impairments in the LHD group could not be easily attributed to domain-specific, linguistic factors.

Although these data are consistent with previously reported WM deficits in the aphasia literature (Baldo & Dronkers, 1999; Beeson et al., 1993; Caspari et al., 1998; Downey et al., 2003), the present results extend these reports by constraining the nature of the WM impairment. That is, it is highly unlikely that the WM deficits of the LHD participants in this study resulted from a primary language impairment, given that systematic manipulation of linguistic complexity had no effect on their WM performance. Furthermore, unlike several previous studies (Baldo & Dronkers, 1999; Beeson et al., 1993), no differences in quality of performance were found between individuals with fluent versus nonfluent aphasia. Rather, all LHD participants were similarly and negatively affected by increasing WM load. The failure to discriminate between aphasia types further supports a domain-general interpretation of these results (Murray et al., 1997a).

## ***1.2 Correlational analyses***

Consistent with the group and WM load effects, LHD subjects' ability to name linguistic stimuli was unassociated with their ability to recall the identical stimuli during n-back tasks; nor was aphasia severity a factor. In fact, the only index of n-back performance related to aphasia severity and picture-naming scores was *Pr face*, the very score designed to measure WM for non-nameable, nonlinguistic stimuli.

It is likely that the association between *Pr face* and aphasia severity reflected simply that face stimuli were more difficult than object stimuli across n-back tasks. A further indication that this relationship was domain-general in nature was that subjects' age, across both LHD and NBD groups, was also significantly related to *Pr face*. That is, more severe aphasia and greater age were both associated with decreased performance for nonlinguistic versus linguistic stimuli, consistent with other reports in the literature. For example, Laures et al. (2003) reported a similar advantage of linguistic over nonlinguistic stimuli for individuals with aphasia during an auditory vigilance task, despite the use of abstract, low-frequency words as linguistic stimuli; Nystrom et al. (2000) found the same effect on an n-back task for NBD subjects. That the LHD group experienced this linguistic advantage to a similar degree as the NBD group demonstrates that, despite their aphasia, they were able to take advantage of an impaired lexical-semantic network to support WM processes. In contrast, the face n-back tasks required relatively pure visual strategies without support from long-term semantic or lexical stores. Evidence from lesion studies and neuroimaging studies suggests distinct networks for the maintenance of novel, complex objects (e.g., faces, scenes, abstract drawings) compared to familiar stimuli, with the former requiring medial temporal lobe activity, including hippocampal areas, as well as PFC activation (Ranganath, 2006). This may reflect top-down feedback to facilitate reconstruction of aspects of novel visual images, given that such images would not correspond to established visual object representations in inferior temporal cortex. Additionally, although care was taken to equate the visual complexity of the nameable and face stimuli by using grayscale photographs, the requirement that face

stimuli be as non-nameable as possible necessitated removing identifying features (e.g., hair) from individual stimuli and equating facial expressions across stimuli. The resulting and unavoidable similarity of faces across successive trials likely made the simple maintenance of low-level visual features insufficient to support accurate WM performance (Hartley et al., 2001), which further increases the possibility that subjects needed to actively construct stable representations of face stimuli during WM trials. Thus, face stimuli are simply more challenging than object stimuli during WM tasks, consistent with the increased RTs for face versus object stimuli in both subject groups and the significant relationships between aphasia severity, age, and WM accuracy for face stimuli.

### ***1.3 Vigilance effects***

Similar to reports by Laures and colleagues (Laures, 2005; Laures et al., 2003), task duration was a factor for LHD but not NBD performance, specifically on the two-back tasks. Unlike Laures (2005), however, this effect was only apparent at the most complex task level and not the 0- or 1-back levels. Similarly, De Renzi and Faglioni (1965) noted that although simple vigilance tests discriminated between brain-damaged versus NBD individuals, restricting the analysis to LHD subjects with versus without aphasia yielded discrimination only under more demanding conditions (i.e., a simple RT vigilance task yielded equal performance). Thus, in line with the overall picture of this study's results, vigilance appeared affected in the LHD subjects only in a load-dependent manner. That true tests of vigilance require at least 30 minutes' duration (Davies & Parasuraman, 1982; Laures et al., 2003) decreases

further the likelihood that these results reflected simply decreased attentional vigilance in aphasia.

Another explanation for the vigilance decrement is that the LHD subjects were more susceptible to *proactive interference* (PI; disrupted memory performance due to the products of internally derived, earlier mental activity; Postle, 2006), leading to decreased n-back performance as they attempted to hold more stimuli on-line during 2-back tasks. However, given the increased inter-stimulus similarity of faces compared to the nameable stimuli, PI should have been most apparent during the face n-back tasks. In fact, the opposite was true: A finer-grained analysis of LHD n-back performance yielded significant vigilance decrements only during the low-frequency 2-back and just trends in the same direction were found for high-frequency and face 2-backs. It remains possible, however, that the non-significant vigilance decrement in the face 2-back condition reflected a floor effect, given that this condition was arguably the most difficult (see Table 5). Regardless, the vigilance decrement of the LHD subjects in this study did not simply reflect impaired sustained attention given the collective results illustrating the specificity of this effect.

#### ***1.4 RT and Br effects***

Overall, RT and Br (bias) effects paralleled accuracy (Pr), further supporting a domain-general locus of influence. No differences were found with respect to word frequency, whereas strong effects were noted for WM load. Whereas the Br signal detection statistic yielded main effects of group, however, RT measures did not. Speed of processing can arguably be viewed as a basic measure of the intactness of

the central nervous system (Hartley et al., 2001). Following Salthouse (1995), WM significantly depends on speed, with undifferentiated decline occurring in older adults. As such, RT was expected to be affected strongly in the aphasia versus control group. Thus, the lack of significant RT differences across groups was an unexpected finding, notwithstanding the non-significant trend in this direction (see Figure 5). These data likely reflected both groups' variable success with speed-accuracy trade-offs. Indeed, correlational analyses of LHD data revealed a significant negative relationship between Pr and RT at the 1-back level: Subjects who responded accurately to 1-back stimuli could also do so more quickly. At the 2-back level, however, the correlation was small and positive ( $r = .16$ ), suggesting that LHD subjects unsuccessfully sacrificed speed for accuracy at this more demanding level (Downey et al., 2004). Alternatively, these data partially support predictions of the general capacity WM model in that subjects' ability to process stimuli more efficiently at the 2-back level did not predict their ultimate WM capacity, as the latter represents the capacity for controlled, executive attention independent of task content (Conway & Engle, 1996).

### ***1.5 WM model predictions***

As noted previously, the crucial distinction between the four primary WM models in the literature concerns what dictates overall capacity, given strong evidence of individual differences (e.g., Daneman & Carpenter, 1980, 1983; Engle, 1999) and disorder-specific capacity effects (e.g., Ellis-Weismer et al., 1999; Tompkins et al., 1994). From the perspective of Baddeley's (1986) multi-component WM model,

these results clearly are inconsistent with problems at the level of the phonological loop (or other linguistic instantiations thereof; Freedman & Martin, 2001). Instead, the domain-general effects in this study provide support for WM impairments at the level of Baddeley's central executive (CE) component. However, that Baddeley (2002; Baddeley et al., 2001) has failed to clearly specify the nature of the CE severely limits the explanatory value of his model for WM in aphasia. Furthermore, these results are collectively inconsistent with predictions of both the resource-sharing (Daneman & Carpenter, 1980, 1983; Just & Carpenter, 1992) and emergent (MacDonald & Christiansen, 2002) views of WM. Regarding the former, WM task performance should be affected in a global manner if *any component* of the task is made more difficult: thus, resource-sharing views predict that increasing linguistic complexity (in this case, via manipulating word frequency) should affect WM performance in a similar manner as increasing WM load. That this study unmistakably differentiated the effects of these manipulations is inconsistent with the notion of task-specific resource demands driving WM. Moreover, that simple RT failed to differentiate the LHD and NBD groups in this study further supports that processing efficiency alone did not drive WM variance, as noted above. Likewise, the emergent view of WM as inextricably tied to the linguistic representations or processes inherent in WM tasks is clearly at odds with the present results. Thus, WM capacity in adults with aphasia appears impaired in a predominantly load- rather than language-dependent manner, consistent with the general capacity view of WM as a domain-free reflection of the ability to control attention (Engle et al., 1999; Hambrick & Engle, 2003; Kane & Engle, 2002).



## 2. A broader picture

### **2.1 Resource theory in aphasia**

This study's results are broadly consistent with the notion of decreased resources or decreased allocation of available resources in individuals with aphasia. However, resource theory is traditionally invoked as a domain-general explanation of *linguistic* performance deficits (Blackwell & Bates, 1995; McNeil, Hula, Matthews, & Doyle, 2004; McNeil et al., 1991; Murray, 1999; Murray & Kean, 2004): Persons with aphasia demonstrate limited resources, inefficient resource allocation, or both *during linguistic tasks* (Shuster, 2004). That this study demonstrated aphasia-related WM impairments in a predominantly load-dependent, domain-general manner is consistent with a growing literature demonstrating fundamentally impaired *nonlinguistic* cognition in aphasia, outside the realm of linguistic task performance (Erickson et al., 1996; Lang, 1989; Laures et al., 2003; Ostergaard & Meudell, 1984; Purdy, 2002; Shallice, 1982; Shisler, 2005).

It is increasingly evident that similar cognitive disorders can and have been identified in virtually every type of brain damage, including right hemisphere impairment (Glosser & Goodglass, 1990; Murray, 2000), traumatic brain injury (Kimberg et al., 1997; Perlstein et al., 2004), multiple sclerosis (Parmenter, Shucard, Benedict, & Shucard, 2006), schizophrenia (Goldberg et al., 2003; Honey & Fletcher, 2006), reading disabilities (Swanson & Alexander, 1997), specific language impairment (Marton & Schwartz, 2003), depression (Harvey et al., 2004), dementia (Baddeley, 2002), Parkinson's disease (Monetta & Pell, 2006), and general effects of aging (Moineau et al., 2005; Salthouse et al., 2003; Smith et al., 2001; Verhaeghen &

Basak, 2005). Thus, results of this study might be better explained by broadening our perspective of the domain-general cognitive disorders that have long been recognized in aphasia. Resource theory as currently conceptualized cannot fully account for such a broad swath of disorder types and loci. Likewise, it is difficult to reconcile such widespread accounts of high-level cognitive impairments across disorders with a prominent role for domain specificity in resulting cognitive impairments.

## ***2.2 Neural connectivity theory***

Such a broad picture has led some researchers to question the specificity of the WM construct itself. For example, in a large study using structural equation modeling to investigate the role of EF in age-related cognitive decline, Salthouse et al. (2003) found that constructs thought to be theoretically separable, including “executive functioning” (i.e., problem-solving tasks), “updating” (i.e., n-back and other WM tasks), and “time-sharing” (i.e., attentional capacity), were highly and positively correlated with a gF factor (i.e., general fluid intelligence or Spearman’s “g”), “implying that nearly the same dimension of individual differences was captured with what are often assumed to be different constructs” (p. 588). The statistical overlap among these constructs has been reflected in theoretical accounts as well. For example, gF has been defined as synonymous with WM (Gray, Chabris, & Braver, 2003), as has inhibition, attention, and EF (Awh et al., 1996; Awh et al., 2006; Engle & Kane, 2004; Keil, 2003). Given their results, Salthouse et al. (2003) cautioned researchers investigating individual cognitive or neuropsychological constructs to recognize that “they are probably studying only one aspect of a larger

phenomenon and that it may be misleading to assume that they have isolated something novel and distinct...” (p. 590).

Salthouse et al. (2003) raised the “intriguing possibility” (p. 590) that the close relationships between WM, attention, and EF reflect a shared dependence on the “integrity of circuits responsible for communication within and across neuroanatomical regions” (p. 590). Given that problems in such communication could be hypothesized to affect the assimilation and synchronization of many different types of processing (Aboitiz et al., 2006), this “neural connectivity hypothesis” (Salthouse et al., 2003, p. 590) has the potential to account at once for the multiplicity of cognitive variables affected by brain damage, and the similarities noted between very different patient populations. That is, the brain should be conceptualized “not as a hierarchy of organized autonomous modules, each of which delivers its output to the next level, but as a set of complex interacting networks that are in a state of dynamic equilibrium with the brain’s environment” (Ramachandran, 2005, p. 372). WM in particular has been characterized as dependent upon reciprocally connected neuronal ensembles, which oscillate synchronously in the service of maintaining active memory during the execution of a cognitive task (Aboitiz et al., 2006). Accordingly, functional neuroimaging studies have found increased WM load to be associated with increased connectivity between frontal, cingulate, and parietal regions; sustained activation of both extrastriate visual and prefrontal cortical areas during memory delays; and increased inter-hemispheric communication between dorsolateral frontal regions (Courtney, Ungerleider, Keil, & Haxby, 1997; Honey & Fletcher, 2006). This idea of a superordinate general ability that is diffusely cortically

represented is far from new; in fact, inspired by principles of equipotentiality and mass action (Lashley, 1929), many researchers have embraced a version of the view that general intelligence (gF) depends on the intactness of the entire cortex, with an injury to any region of the brain resulting in intellectual decrement, above and beyond the contribution of subordinate, domain-specific factors (e.g., Basso et al., 1981; Basso et al., 1973).

Plausible sources of decreased connectivity or inefficient neural communication following brain damage (or aging) include variations in neuronal quantity or density, neurotransmitter quantity or balance, synaptic density, or degree of myelination (Salthouse et al., 2003). Given the known cascade of neural events following brain damage (Heiss & Thiel, 2006; Leker & Shohami, 2002; Neumar, 2000), it is likely that a combination of these sources may lead to pervasive effects of localized brain damage on neural connectivity both adjacent to the original insult and in remote cortical areas (Heiss & Thiel, 2006). In fact, it is well known that aphasia, regardless of size or site of lesion, is associated with a common hypometabolism in the left temporal and temporoparietal regions (Karbe et al., 1998), providing evidence for generally decreased left-hemisphere connectivity post-insult (McNeil, Hula, Matthews, & Doyle, 2004). Accordingly, a growing number of researchers are exploring non-linguistic cognitive treatments for aphasia (Coelho, 2005; Fischler, 2000; Hardin & Ramsberger, 2004; Helm-Estabrooks, 1998, 2002; Helm-Estabrooks & Albert, 1991; Helm-Estabrooks, Connor, & Albert, 2000; Mayer & Murray, 2002; Murray, 1999, 2003; Ramsberger, 2005).

## 2.4 Summary

In sum, these results and literature review support that a damaged brain does not work like an intact brain and cannot be conceptualized as simply a brain with one or more “pieces” missing or damaged (cf. Raymer & Gonzalez-Rothi, 2001). Instead, evidence has shown that a damaged brain functions in quantitatively and qualitatively different ways than an intact brain, and it is this pervasive change in functioning that is posited to account for domain-general, persistent changes in broad functions such as WM following a left-hemisphere, aphasia-causing stroke. This conclusion in no way denies the obvious fact that the brain is organized specifically according to certain abilities. However, as a consequence of brain damage, aphasia is inextricably associated with additional, higher-level deficits that are:

- 1) *domain-general*: cutting across domains and modalities beyond language
- 2) *common to most types of brain damage*: due to massive neural interconnectivity in normal high-level cognitive functioning and widespread disturbance of this connectivity following damage.
- 3) *negatively associated with language function*: these domain-general deficits are not the primary cause of language dysfunction in aphasia, given the lack of frank aphasia in other types of brain damage. However, such deficits exacerbate language-based impairments and negatively affect treatment outcomes.

### 3. The remaining case for domain specificity: Language-related WM influences

Although these data strongly support domain-general WM impairments in aphasia, the fact that the rejection of domain-specific influences rests in part on simply the absence of an effect (i.e., word frequency as a WM modulator) necessitates that alternative interpretations be considered. Therefore, the following section explores the remaining case for WM domain-specificity in aphasia.

#### **3. 1 *Low versus high-frequency object names***

Across LHD and NBD groups, there were no measurable differences in accuracy (PR), RT, or bias (BR) between n-back tasks with low- versus high-frequency word stimuli. A possible interpretation of these data calls for a closer look at specific task demands, according to recent connectionist theories of lexical access (Bock & Levelt, 1994; Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Levelt, 1999; Levelt, Roelofs, & Meyer, 1999). According to these theories, lexical knowledge is embedded in a network consisting of two or more layers: for example, a semantic layer (i.e., conceptual level), a “word” or lemma layer (i.e., syntactic category information), and a phoneme or “lexeme” layer (i.e., word form or sound level; Dell et al., 1997; Levelt et al., 1999). Although the models differ in terms of how these levels interact (e.g., activation flow in one direction, Levelt, 1999; Levelt et al., 1999; vs. bidirectional activation between layers, Dell et al., 1997), they are in general agreement that the information within layers forms relatively *independent* levels of representation (Caramazza, 1997). That is, lexical-semantic activation does not necessarily imply modality-specific lexical-phonological activation during word

retrieval, depending on potential interactions amongst representations at different levels and the nature of the task at hand (Bock & Levelt, 1994; Pashek & Tompkins, 2002).

Therefore, although task formats in this study were chosen specifically to minimize overt verbal output, given the problems of confounding language with general cognitive resources identified in previous WM and aphasia studies (Caspari et al., 1998; Tompkins et al., 1994), it is possible that this design minimized n-back word frequency effects by requiring retrieval at the conceptual and/or lemma level, rather than the lexeme (word form) level that is obligatory during, for example, confrontation-naming tasks. This possibility may be interpreted in three ways. First, many theories of the locus of the word frequency effect place this variable at the *lexeme* level (Bock & Levelt, 1994; Caramazza, 1997; Luzzatti et al., 2002); therefore, during tasks which do not require lexical retrieval at this level, word frequency might not be expected to affect performance. Second, consistent with a resource-based perspective, fewer *linguistic resources* are required for lemma versus lexeme retrieval: This, then, would account for expected effects of word frequency manipulations during the picture naming task (i.e., lexeme retrieval), with insufficient taxing of linguistic resources during the n-back task (i.e., lemma retrieval). Third, given the time-limited nature of lexical retrieval in connectionist models (i.e., spreading activation amongst levels constrains how quickly retrieval processes may proceed), it may be that the rapid n-back time-course did not allow for word-related activation to spread from the lemma to lexeme levels in time for the word frequency

effect to take place. Considering the rapid pace at which normal communication and conversation take place, however, evidence for the latter possibility is limited.

On the flip side, the purely psycholinguistic concepts of the lexeme and lemma become blurred when neurophysiological data are considered. Functional neuroimaging evidence for WM-related storage and rehearsal processes in the PFC demonstrates that the very regions proposed to mediate phonological, grammatical, and articulatory aspects of speech and language (e.g., premotor cortex, SMA, Broca's area) are active during WM tasks during which no overt responses are required (Smith & Jonides, 1999). Simple item recognition tasks (with letter, face, or meaningless object (e.g., scrambled faces) stimuli), compared with control tasks result in left Broca's area, SMA, and premotor cortex activation (Awh et al., 1996; Courtney et al., 1997). Moreover, the activation in Broca's area closely matches that obtained during explicit phonological tasks such as rhyme judgment (Smith et al., 1998). Similar activation patterns have been obtained in fMRI and PET studies using 2- and 3-back tasks (Cohen et al., 1997). This activation disappears when the n-back tasks are subtracted from a control task in which participants are explicitly instructed to rehearse each letter silently, indicating that similar rehearsal mechanisms are utilized for item recognition, rhyme judgment, and n-back tasks. Therefore, "frontal regions that no doubt evolved for the purpose of spoken language appear to be recruited to keep verbal information active in working memory" (Smith & Jonides, 1999, p. 1658). That is, neuroimaging data support that phonological rehearsal is a critical component of WM and is instantiated in a similar manner whether the WM task requires covert or overt verbal rehearsal (i.e., lexical retrieval). The processes



ascribed to activated areas closely match lexeme-type processes described by connectionist lexical retrieval theories.

If then, during n-back tasks, NBD subjects clearly activate brain areas that mediate the very aspects of language processing affected by word frequency, why did this variable fail to affect LHD or NBD WM performance? One possibility is that the LHD subjects approached the n-back task in a qualitatively different manner than the NBD subjects from whom neurophysiological WM data are most often collected. However, it is also possible that phonological recoding processes activated automatically in healthy adults faced with visual stimuli (Postle, 2006) are sufficient but not *vital* for successful n-back performance under certain conditions. N-back task structure disallows formally separating out the integrity of the phonological loop (Mueller et al., 2003), and in this case, that the linguistic stimuli were all recognizable, common objects may have encouraged semantic recoding strategies to support n-back performance.

### ***3.2 Domain-specific interpretations***

The current results are not completely inconsistent with Caplan, Waters, and colleagues (Caplan & Waters, 1995, 1999; Rochon, Waters, & Caplan, 2000), who argued that the WM system used for purely linguistic processing is entirely separate from the WM system used for complex, verbally mediated tasks. According to this view, increasing the component complexity of a given WM task (i.e., increasing domain-specific resource demands) should not correlate with overall WM capacity as typically measured by span tasks, because the two types of processing are controlled

by distinct cognitive systems. On the other hand, it is unclear how this theory would account for the spectrum of results in this study and in other studies which clearly demonstrate nonlinguistic cognitive deficits in adults with aphasia or with other types and loci of brain damage. That is, if aphasia represents an impairment of a domain-specific, interpretive WM processor (Caplan & Waters, 1999), would the “post-interpretive” deficits found in this and other studies of WM in aphasia be conceptualized as representing impairment to a separate cognitive module? In that case, many patients with aphasia would suffer from dual, modular WM deficits: this seems a serious threat to theoretical parsimony (Christiansen & MacDonald, 1999) and to what is known about functional neural architecture (Friston et al., 1996; Smith & Jonides, 1997). A more plausible rendition of Caplan and Waters’ (1999) dual WM architecture was given by Gibson and Roberts (1999), in which a domain-general WM system is conceptualized as entirely distinct from the linguistic (non-WM) processing system. Given that the domain-general WM construct might actually represent the overlap between many aspects of cognition relying upon reciprocal communication amongst complex neuronal circuits (Aboitiz et al., 2006; Salthouse et al., 2003), this view differs little from the domain-general conclusion derived above. That is, aphasia represents an impairment of a language-specific area of the brain, but as a result of the aphasia-causing damage, the brain of such an individual functions in a quantitatively and qualitatively different manner than that of a healthy person, leading to the spectrum of higher-level cognitive problems that are apparent across a wide range of neurogenic disorders.

#### 4. Shortcomings and future research

One might argue that the results of this study, rather than providing evidence towards a domain-general impairment overlaid on the language impairment in aphasia, simply reflect: 1) inadequate power to yield a statistically significant effect of language complexity (i.e., word frequency) during the n-back tasks; 2) inadequacy of the n-back task for measuring WM; 3) insufficiency of word frequency to represent linguistic complexity; or 4) unfalsifiability of the domain-general construct. Systematic consideration of each of these possibilities follows.

##### ***4.1 Power and effect size***

Clearly the total number of participants with aphasia was comparatively small. An examination of effect sizes, however, indicates that the current results do not demonstrate merely a lack of statistical power. According to Cohen (1977), partial eta squared ( $\eta_p^2$ ) = .01 represents a small effect size,  $\eta_p^2$  = .06 corresponds to a medium effect size, and  $\eta_p^2$  = .14 is a large effect size. Limiting the focus to the LHD group, analyses revealed  $\eta_p^2$  = .02 for the effect of language load (i.e., high vs. low frequency words), whereas  $\eta_p^2$  = .89 for the effect of WM load. It is highly unlikely that further subject recruitment would change the nature of these results, given the considerable difference in the strength of these effects.

##### ***4.2 N-back task***

Although a key strength of the n-back paradigm is the opportunity for parametric manipulation of WM load within a single task, several researchers have

questioned whether the  $n$ -back task represents WM at lower levels of  $n$  (Harvey et al., 2004; Jarrold & Towse, 2006; Salthouse et al., 2003; Verhaeghen & Basak, 2005). Instead, the 0- or 1-back tasks might be classified as maintenance tasks, with only 2- and 3-back tasks classified more specifically as WM tasks (i.e., involving manipulation or executive processes as well as maintenance of stimuli; Harvey et al., 2004; Nystrom et al., 2000). In fact, during functional neuroimaging studies of  $n$ -back tasks with NBD subjects, activation in DLPFC has increased in a step-wise fashion: There is nominal difference between DLPFC activation in 0- versus 1-back tasks, and a large increase from 1- to 2-back tasks (Cohen et al., 1997; Smith et al., 1998). The current results are broadly consistent with this interpretation of the  $n$ -back task, given the high correlation between accuracy scores on the 0- and 1-back tasks, and the much lower correlations between 0- or 1-back performance with 2-back accuracy (Table 7). It is notable, however, that post-hoc analyses of LHD group performance demonstrated significant changes when moving from both the 0- to 1-back as well as 1- to 2-back levels. Likewise, others have found the 1-back task useful due to unacceptably high error rates with higher levels of  $n$ , similar to the current study, especially with older adults (Hartley et al., 2001), and the 1-back task has been characterized as a “pure” measure of the ability to update continuously memory contents (Harvey et al., 2004). Thus, although the 0-back task is designed primarily as a measure of sustained attention, it is likely that in brain-damaged or aging populations the 1-back as well as 2-back tasks fulfill the requirements of a WM-demanding task.

Jarrold and Towse (2006) questioned whether the continuous updating assumed to take place during an n-back task “corresponds to the kind of *active* processing that is central to other tasks assumed to tap working memory” (p. 40). In contrast, Postle (2006) noted the complexity of the n-back task for making inferences about neuroanatomical bases of varying memory load, consistent with previous suggestions that successful n-back performance represents executive functions such as attention and monitoring (Watter et al., 2001). These divergent views likely reflect the complexity of the WM construct. Likewise, a superficial search of the literature yields considerable criticism leveled at just about any existing WM, attention, or EF measure (e.g., Salthouse et al., 2003; Shuster, 2004). A possible solution for future studies of WM in aphasia is using multiple WM measures to overcome the drawbacks inherent in individual WM tasks, including techniques such as structural equation modeling to pull apart critical WM variables from unrelated task variance (Miyake et al., 2000; Salthouse et al., 2003).

#### ***4.3 Linguistic complexity parameters***

Word frequency clearly represents linguistic complexity across a wide variety of lexical tasks. This parameter was chosen to define language complexity in this study following systematic consideration of other, less potent factors affecting lexical recognition, recall, and production (Hulme et al., 1995; Jusczyk, 1997; Mainela-Arnold & Evans, 1995). As in the literature, word frequency yielded effects in the expected direction (high > low) during confrontation naming of stimuli for the LHD group. Therefore, that this parameter failed to yield WM differences within and

between LHD and NBD groups strongly supports that domain-general rather than domain-specific factors drive individual differences in WM.

Alternatively, linguistic influences on WM function in adults with aphasia may simply not be measurable in terms of word frequency in the n-back task. Many other possible instantiations of linguistic complexity exist, and it is possible that traditional WM tasks historically have not taxed syntactic and semantic processing systems enough to gauge the true influence of these variables (Crosson et al., 1999; N. Martin, Saffran, & Dell, 1996; R. C. Martin & Feher, 1990). Accordingly, Baddeley (2000) recently acknowledged multi-modal representations in WM by creating the “episodic buffer,” implying that linguistic properties outside of phonology might also affect WM-based rehearsal functions (Mueller et al., 2003). In fact, semantic knowledge has been shown to support lexical retrieval (Pexman, Lupker, & Hino, 2002) and verbal short-term storage (Collette et al., 2001), with larger verbal STM span performance for words than non-words (Hulme et al., 1995), and additional activation regions during verbal versus non-verbal STM tasks likely associated with supporting lexical and semantic language representations (Collette et al., 2001). Therefore, given that this study is the first in the aphasia literature in which linguistic complexity has been addressed and systematically manipulated to test WM, it is indisputable that future research should test additional sources for linguistic WM influences. Possible candidates include parameters such as semantic typicality (Rosch, 1975) which may act on earlier processing levels than word frequency (Dell et al., 1997).

#### 4.4 “Testing” domain generality

Tompkins et al. (1994) cautioned against a major risk in invoking a domain-general, resource-based perspective to explicate performance deficits: “it can be too seductive” (p. 911). Shuster (2004) similarly argued that the concept is not falsifiable and can be applied to any data. A similar problem has been raised for the concept of domain-specificity, however, in that the number of putative processing systems cannot be constrained with such theories (MacDonald & Christiansen, 2002). Thus, neither standpoint is immune from serious methodological problems when invoked post-hoc.

To counteract this risk, Tompkins et al. (1994) suggested formulating and testing *specific predictions* in pursuing a resource-based explanatory focus. The current study represented an attempt to do just that: Rather than testing whether or not resource theory can explain aphasia, we explored specifically whether *linguistic* phenomena might influence performance on a traditionally cognitive or nonlinguistic task. That the linguistic manipulations entertained herein did not appear to alter the performance of individuals with aphasia does not ultimately prove resource theory in the psychological realm nor neural connectivity hypotheses in the neurobiological realm. Instead, these data provide another piece of evidence towards the growing realization that aphasia symptomology cannot be explained on a purely linguistic basis. Accumulation of evidence of this type is the foundation on which complex scientific theories are formed (McNeil et al., 2004).

#### ***4.5 Future Research***

Results of this study revealed that previous research demonstrating verbal WM impairments in patients with aphasia represents just one piece of a larger phenomenon that will require a broader perspective to fully appreciate. Therefore, future research toward elucidating the domain-general versus domain-specific contributions to cognitive impairments in aphasia should include the following: 1) participants with different types and locations of brain damage (e.g., RHD, TBI); 2) systematic manipulation of additional linguistic parameters; for example, semantic typicality (Rosch, 1975); 3) systematic comparisons between different types of nonverbal stimuli: for example, spatial versus non-spatial WM, given the known dissociations between these types of memory in healthy adults (Postle, 2006; Shah & Miyake, 1996); 4) different types or a combination of WM tasks, given the known problems with individual measures of such a complex construct; and 5) continued exploration of treatment options for cognitive impairments across disorder groups.



## References:

- Aboitiz, F., Garcia, R. R., Bosman, C., & Brunetti, E. (2006). Cortical memory mechanisms and language origins. *Brain and Language*, 98, 40-56.
- Adcock, R. A., Constable, R. T., Gore, J. C., & Goldman-Rakic, P. S. (2000). Functional neuroanatomy of executive processes involved in dual-task performance. *Proceedings of the National Academy of Sciences*, 97(7), 3567-3572.
- Alexander, M. P., & Stuss, D. T. (2006). Frontal injury: Impairments of fundamental processes lead to functional consequences. *Journal of the International Neuropsychological Society*, 12, 192-193.
- Attneave, R., & Arnoult, M. D. (1956). The quantitative study of shape and pattern perception. *Psychological Bulletin*, 53(6), 452-471.
- Awh, E., Jonides, J., Smith, E. E., Schumacher, E. H., Koeppe, R. A., & Katz, S. (1996). Dissociation of storage and rehearsal in verbal working memory: Evidence from Positron Emission Tomography. *Psychological Science*, 7(1), 25-31.
- Awh, E., Vogel, E. K., & Oh, S.-H. (2006). Interactions between attention and working memory. *Neuroscience*, 139, 201-206.
- Baddeley, A. D. (1986). *Working memory*. New York: Oxford University Press.
- Baddeley, A. D. (2000). The episodic buffer: A new component of working memory? *Trends in Cognitive Sciences*, 4(11), 417-423.
- Baddeley, A. D. (2002). Is working memory still working? *European Psychologist*, 7(2), 85-97.

- Baddeley, A. D., Chincotta, D., & Adlam, A. (2001). Working memory and the control of action: Evidence from task switching. *Journal of Experimental Psychology: General*, 130(4), 641-657.
- Baddeley, A. D., Gathercole, S., & Papagno, C. (1998). The phonological loop as a language learning device. *Psychological Review*, 105(1), 158-173.
- Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. A. Bower (Ed.), *The psychology of learning and motivation: Advances in research and theory* (Vol. 8). Philadelphia: Academic Press.
- Baddeley, A. D., & Hitch, G. J. (1994). Developments in the concept of working memory. *Neuropsychology*, 8(4), 485-493.
- Baddeley, A. D., Thomson, N., & Buchanan, M. (1975). Word length and the structure of short-term memory. *Journal of Verbal Learning and Verbal Behavior*, 14, 575-589.
- Bailey, S., Powell, G. E., & Clark, E. (1981). A note on intelligence and recovery from aphasia: the relationship between Raven's Matrices scores and change on the Schuell Aphasia Test. *British Journal of Disorders of Communication*, 16(3), 193-203.
- Baldo, J. V., & Dronkers, N. F. (1999). Verbal and nonverbal short-term memory in patients with conduction aphasia and prefrontal cortex lesions. *Brain and Language*, 69, 475-478.
- Baldo, J. V., Dronkers, N. F., Wilkins, D. P., Ludy, C., Raskin, P., & Kim, J. (2004). Is problem solving dependent on language? *Brain and Language*, 92, 240-250.

- Baldo, J. V., Elder, J. T., Larsen, J., Dronkers, N. F., Redfern, B., & Ludy, C. (2001). Is cognition intact in patients with aphasia? *Brain and Language*, 79, 64-67.
- Basso, A., Capitani, E., Luzzatti, C., & Spinnler, H. (1981). Intelligence and left hemisphere disease: the role of aphasia, apraxia and size of lesion. *Brain*, 104, 721-734.
- Basso, A., Capitani, E., Luzzatti, C., Spinnler, H., & Zanobio, M. E. (1985). Different basic components in the performance of Broca's and Wernicke's aphasics on the colour-figure matching test. *Neuropsychologia*, 23(1), 51-59.
- Basso, A., De Renzi, E., Faglioni, P., Scotti, G., & Spinnler, H. (1973). Neuropsychological evidence for the existence of cerebral areas critical to the performance of intelligence tasks. *Brain*, 96, 715-728.
- Bayles, K. A., & Tomoeda, C. S. (1993). *Arizona Battery for Communication Disorders of Dementia*. Tuscon, AZ: Canyonlands Publishing.
- Bayliss, D. M., Jarrold, C., Gunn, D. M., & Baddeley, A. D. (2003). The complexities of complex span: Explaining individual differences in working memory in children and adults. *Journal of Experimental Psychology: General*, 132(1), 71-92.
- Beeson, P. M., Bayles, K., & Kaszniak, A. W. (1993). Memory impairment and executive control in individuals with stroke-induced aphasia. *Brain and Language*, 45, 253-275.
- Blackwell, A., & Bates, E. (1995). Inducing agrammatic profiles in normals: evidence for the selective vulnerability of morphology under cognitive resource limitation. *Journal of Cognitive Neuroscience*, 7(2), 228-257.

- Bock, K., & Levelt, W. J. M. (1994). Language production: Grammatical encoding. In M. Gernsbacher (Ed.), *Handbook of Psycholinguistics* (pp. 945-983). Philadelphia: Academic Press.
- Bonatti, L. (2006). Pyscope X. 2006, from <http://psy.ck.sissa.it/>
- Borod, J. C., Carper, M., & Goodglass, H. (1982). WAIS Performance IQ in aphasia as a function of auditory comprehension and constructional apraxia. *Cortex*, 18, 199-210.
- Brown, L., Sherbenou, R. J., & Johnsen, S. K. (1997). *Test of Nonverbal Intelligence: Third Edition*. London: Pearson Assessments.
- Buckingham, H. W. (1985). Perseveration in aphasia. In S. Newman & R. Epstein (Eds.), *Current perspectives in dysphasia* (pp. 113-154). London: Churchill Livingstone.
- Burgio, F., & Basso, A. (1997). Memory and aphasia. *Neuropsychologia*, 35(6), 759-766.
- Callicott, J. H., Mattay, V. S., Bertolino, A., Finn, K., Coppola, R., Frank, J. A., et al. (1999). Physiological characteristics of capacity constraints in working memory as revealed by functional MRI. *Cerebral Cortex*, 9, 20-26.
- Caplan, D., & Waters, G. S. (1995). Aphasic disorders of syntactic comprehension and working memory capacity. *Cognitive Neuropsychology*, 12(6), 637-649.
- Caplan, D., & Waters, G. S. (1996). Syntactic processing in sentence comprehension under dual-task conditions in aphasic patients. *Language and Cognitive Processes*, 11, 525-551.

- Caplan, D., & Waters, G. S. (1999). Verbal working memory and sentence comprehension. *Behavioral and Brain Sciences*, 22, 77-126.
- Caramazza, A. (1997). How many levels of processing are there in lexical access? *Cognitive Neuropsychology*, 14(1), 177-208.
- Caspari, I., Parkinson, S. R., LaPointe, L. L., & Katz, R. C. (1998). Working memory and aphasia. *Brain and Cognition*, 37, 205-223.
- Chapey, R. (2001). Cognitive stimulation: Stimulation of recognition/comprehension, memory, and convergent, divergent, and evaluative thinking. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (pp. 397-434). Philadelphia: Lippincott Williams & Wilkins.
- Chapey, R., & Hallowell, B. (2001). Introduction to language intervention strategies in adult aphasia. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (pp. 3-17). Philadelphia: Lippincott Williams & Wilkins.
- Chapey, R., Rigrodsky, S., & Morison, E. M. (1977). Aphasia: a divergent semantic interpretation. *Journal of Speech and Hearing Disorders*, 42, 287-295.
- Christiansen, M. H., & MacDonald, A. W. (1999). Fractionated working memory: Even in pebbles, it's still a soup stone. *Behavioral and Brain Sciences*, 22(1), 97-98.
- Cicerone, K. D. (2002). Remediation of "working attention" in mild traumatic brain injury. *Brain Injury*, 16(3), 185-195.

- Coelho, C. A. (2005). Direct attention training as a treatment for reading impairment in mild aphasia. *Aphasiology*, 19(3/4/5), 275-283.
- Cohen, J. D. (1977). *Statistical power analysis for the behavioral sciences, revised edition*. New York: Academic Press.
- Cohen, J. D., Forman, S. D., Braver, T. S., Casey, B. J., Servan-Schreiber, D., & Noll, D. C. (1994). Activation of the prefrontal cortex in a nonspatial working memory task with functional MRI. *Human Brain Mapping*, 1, 293-304.
- Cohen, J. D., Perlstein, W. M., Braver, T. S., Nystrom, L. E., Noll, D. C., Jonides, J., et al. (1997). Temporal dynamics of brain activation during a working memory task. *Nature*, 386, 604-608.
- Collette, F., Majerus, S., Van der Linden, M., Dabe, P., Degueldre, C., Delfiore, G., et al. (2001). Contribution of lexico-semantic processes to verbal short-term memory tasks: A PET activation study. *Memory*, 9(4/5/6), 249-259.
- Coltheart, M. (1981). The MRC Psycholinguistic Database. *Quarterly Journal of Experimental Psychology*, 33A, 497-505.
- Conway, A. R. A., & Engle, R. W. (1996). Individual differences in working memory capacity: More evidence for a general capacity theory. *Memory*, 4(6), 577-590.
- Courtney, S. M. (2004). Attention and cognitive control as emergent properties of information representation in working memory. *Cognitive, Affective, & Behavioral Neuroscience*, 4(4), 501-516.

- Courtney, S. M., Ungerleider, L. G., Keil, K., & Haxby, J. V. (1997). Transient and sustained activity in a distributed neural system for human working memory. *Nature*, 386(6625), 608-611.
- Cowan, N. (1998). Visual and auditory working memory capacity. *Trends in Cognitive Sciences*, 2(3), 77-78.
- Crawford, J. R. (1998). Introduction to the assessment of attention and executive functioning. *Neuropsychological Rehabilitation*, 8(3), 209-211.
- Crosson, B., Rao, S. M., Woodley, S. J., Rosen, A. C., Bobholz, J. A., Mayer, A., et al. (1999). Mapping of semantic, phonological, and orthographic verbal working memory in normal adults with functional magnetic resonance imaging. *Neuropsychology*, 13(2), 171-187.
- Davies, D., & Parasuraman, R. (1982). *The psychology of vigilance*. London: Academic Press, Inc.
- De Renzi, E., & Faglioni, P. (1965). The comparative efficacy of intelligence and vigilance tests in detecting hemispheric cerebral damage. *Cortex*, 1, 410-433.
- De Renzi, E., & Vignolo, L. A. (1962). The Token Test: A sensitive test to detect receptive disturbances in aphasics. *Brain*, 85, 665-678.
- Dehaene, S., & Changeux, J. P. (1991). The Wisconsin Card Sorting Test: theoretical analysis and modeling in a neural network. *Cerebral Cortex*, 1(1), 62-79.
- Dell, G. S., Schwartz, M. F., Martin, N., Saffran, E. M., & Gagnon, D. A. (1997). Lexical access in aphasic and nonaphasic speakers. *Psychological Review*, 104(4), 801-838.

- Denckla, M. B. (1996). A theory and model of executive function: a neuropsychological perspective. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 263-278). Baltimore, MD: Paul H. Brookes Publishing Co.
- Dollaghan, C., Biber, M., & Campbell, T. (1993). Lexical influences on nonword repetition. *Applied Psycholinguistics*, 16, 211-222.
- Dominey, P. F., Hoen, M., Blanc, J.-M., & Lelekov-Boissard, T. (2003). Neurological basis of language and sequential cognition: Evidence from simulation, aphasia, and ERP studies. *Brain and Language*, 86, 207-225.
- Downey, R. A., Wright, H. H., Schwartz, R. G., Newhoff, M., Love, T., & Shapiro, L. (2003). *Toward a measure of working memory in aphasia*. Paper presented at the American Speech-Language-Hearing Association.
- Dugbartey, A. T., Rosenbaum, J. G., Sanchez, P. N., & Townes, B. D. (1999). Neuropsychological assessment of executive functions. *Seminars in Clinical Neuropsychiatry*, 4(1), 5-12.
- Dunbar, K., & Sussman, D. (1995). Toward a cognitive account of frontal lobe function: simulating frontal lobe deficits in normal subjects. *Annals of the New York Academy of Sciences*, 769, 289-304.
- Duncan, J., Burgess, P. W., & Emslie, H. (1995). Fluid intelligence after frontal lobe lesions. *Neuropsychologia*, 33(3), 261-268.
- Dunn, O. J. (1961). Multiple comparisons among means. *Journal of the American Statistical Association*, 56, 52-64.



- Edwards, S., Ellams, J., & Thompson, J. (1976). Language and intelligence in dysphasia: Are they related? *British Journal of Disorders of Communication*, 11(2), 83-94.
- Ekman, P., & Friesen, W. (Artist). (1976). *Pictures of facial affect*
- Ellis-Weismer, S., Evans, J., & Hesketh, L. J. (1999). An examination of verbal working memory capacity in children with Specific Language Impairment. *Journal of Speech, Language, and Hearing Research*, 42, 1249-1260.
- Engle, R. W. (2002). Working memory capacity as executive attention. *Current Directions in Psychological Science*, 11(1), 19-23.
- Engle, R. W., & Kane, M. J. (2004). Executive attention, working memory capacity, and a two-factor theory of cognitive control. *The Psychology of Learning and Motivation*, 44, 145-199.
- Engle, R. W., Kane, M. J., & Tuholski, S. W. (1999). Individual differences in working memory capacity and what they tell us about controlled attention, general fluid intelligence, and functions of the prefrontal cortex. In A. Miyake & P. Shah (Eds.), *Models of working memory* (pp. 102-134). Cambridge, UK: Cambridge University Press.
- Engle, R. W., Nations, J. K., & Cantor, J. (1990). Is "working memory capacity" just another name for word knowledge? *Journal of Educational Psychology*, 82(4), 799-804.
- Erickson, R. J., Goldinger, S., & LaPointe, L. L. (1996). Auditory vigilance in aphasic individuals: Detecting non-linguistic stimuli with full or divided attention. *Brain and Cognition*, 30, 244-253.

- Evans, J., Coady, J., Sizemore, M., & Mainela-Arnold, E. (in prep). Rapid Automatic Naming in children with SLI: The impact of phonotactic probability and neighborhood density.
- Evans, J., Turkstra, L., & Pollak, S. (in prep). Working memory for emotions in adolescents with and without language impairments.
- Fan, J., Fossella, J., Sommer, T., Wu, Y., & Posner, M. I. (2003). Mapping the genetic variation of executive attention onto brain activity. *Proceedings of the National Academy of Sciences*, 100(12), 7406-7411.
- Feldman-Barrett, L., Tugade, M. M., & Engle, R. W. (2004). Individual differences in working memory capacity and dual-process theories of the mind. *Psychological Bulletin*, 130(4), 553-573.
- Fischler, I. (2000). Attention, resource allocation, and language. In S. E. Nadeau, L. J. Gonzalez-Rothi & B. Crosson (Eds.), *Aphasia and language: Theory to practice* (pp. 348-371). New York: Guildford Press.
- Fratalli, C. M., Thompson, C. K., Holland, A. L., Wohl, C. B., & Ferketic, M. M. (1995). *The American Speech-Language-Hearing Association Functional Assessment of Communication for Adults*. Rockville, MD: ASHA.
- Freedman, M. L., & Martin, R. C. (2001). Dissociable components of short-term memory and their relation to long-term learning. *Cognitive Neuropsychology*, 18(3), 193-226.
- Friedmann, N., & Gvion, A. (2003). Sentence comprehension and working memory limitation in aphasia: A dissociation between semantic-syntactic and phonological reactivation. *Brain and Language*, 86, 23-39.

- Friston, K. J., Price, C. J., Fletcher, P. C., Moore, C., Frackowiak, R. S. J., & Dolan, R. J. (1996). The trouble with cognitive subtraction. *NeuroImage*, 4, 97-104.
- Fuster, J. M. (1973). Unit activity in prefrontal cortex during delayed-response performance: Neural correlates of transient memory. *Journal of Neurophysiology*, 36, 61-78.
- Gainotti, G., Caltagirone, C., & Miceli, G. (1977). Poor performance of right brain-damaged patients on Raven's Coloured Progressive Matrices: derangement of general intelligence or of specific abilities? *Neuropsychologia*, 15, 675-680.
- Gainotti, G., Carlomagno, S., Craca, A., & Silveri, M. C. (1986). Disorders of classificatory activity in aphasia. *Brain and Language*, 28, 181-195.
- Gallagher, R. E. (1994). Assessing cognitive functions in severe aphasia: neuropsychological examination. *Seminars in Speech and Language*, 15(1), 17-36.
- Gibbs, S., & D'Esposito, M. (2006). A functional magnetic resonance imaging study of the effects of pergolide, a dopamine receptor agonist, on component processes of working memory. *Neuroscience*, 139, 359-371.
- Gibson, E., & Roberts, R. (1999). Interpretive and post-interpretive processes in sentence comprehension. *Behavioral and Brain Sciences*, 22(1), 100-101.
- Glogau, S., Ellgring, H., Elger, C. E., & Helmstaedter, C. (2003). Face and facial expression memory in temporal lobe epilepsy patients: preliminary results. *Epilepsy & Behavior*, 5, 106-112.

- Glosser, G., & Goodglass, H. (1990). Disorders in executive control functions among aphasic and other brain-damaged patients. *Journal of Clinical and Experimental Neuropsychology*, 12(4), 485-501.
- Goldberg, T. E., Egan, M. F., Gscheidle, T., Coppola, R., Weickert, T., Kolachana, B. S., et al. (2003). Executive subprocesses in working memory: Relationship to catechol-O-methyltransferase Val158Met genotype and schizophrenia. *Archives of General Psychiatry*, 60(9), 889-896.
- Goldenberg, G., Dettmers, H., Grothe, C., & Spatt, J. (1994). Influence of linguistic and non-linguistic capacities on spontaneous recovery of aphasia and on success of language therapy. *Aphasiology*, 8(5), 443-456.
- Goldman-Rakic, P. S. (1987). Circuitry of the prefrontal cortex and the regulation of behavior by representational memory. In V. B. Mountcastle, F. Plum & S. R. Geiger (Eds.), *Handbook of neurobiology* (pp. 373-417). Bethesda: American Physiological Society.
- Goldman-Rakic, P. S. (1993). Specification of higher cortical functions. *Journal of Head Trauma Rehabilitation*, 8(1), 13-23.
- Goldstein, K. (1948). *Language and language disturbances*. New York: Grune & Stratton.
- Goldstein, K., & Scheerer, M. (1941). Abstract and concrete behavior: An experimental study with special tests. *Psychological Monographs*, 53(2).
- Gray, J. R., Chabris, C. F., & Braver, T. S. (2003). Neural mechanisms of general fluid intelligence. *Nature Neuroscience*, 6(3), 316-322.

- Grigoriou, M., & Mihailescu, L. (1979). Operational strategies in the thinking of aphasics and patients with right hemisphere lesions. *Neurologie et Psychiatrie*, 17(4), 293-302.
- Gutbrod, K., Cohen, R., Mager, B., & Meier, E. (1989). Coding and recall of categorized material in aphasics. *Journal of Clinical and Experimental Neuropsychology*, 11(6), 821-841.
- Haarmann, H. J., Just, M. A., & Carpenter, P. A. (1997). Aphasic sentence comprehension as a resource deficit: A computational approach. *Brain and Language*, 59, 76-120.
- Hambrick, D. Z., & Engle, R. W. (2003). The role of working memory in problem solving. In J. E. Davidson & R. J. Sternberg (Eds.), *The psychology of problem solving* (pp. 176-206). London: Cambridge Press.
- Hamsher, K. (1998). Intelligence and aphasia. In M. T. Sarno (Ed.), *Acquired aphasia* (Third ed., pp. 341-373). San Diego, CA: Academic Press.
- Hardin, K., & Ramsberger, G. (2004). *Treatment of attention in aphasia*. Paper presented at the Clinical Aphasiology Conference, Park City, Utah.
- Hartley, A. A., Speer, N. K., Jonides, J., Reuter-Lorenz, P., & Smith, E. E. (2001). Is the dissociability of working memory systems for name identity, visual object identity, and spatial location maintained in old age? . *Neuropsychology*, 15(1), 3-17.
- Harvey, P. O., Bastard, G. L., Pochon, J. B., Levy, R., Allilaire, J. F., Dubois, B., et al. (2004). Executive functions and updating of the contents of working memory in unipolar depression. *Journal of Psychiatric Research*, 38, 567-576.

- Haxby, J. V., Ungerleider, L. G., Horwitz, B., Maisog, J. M., Rapoport, S. I., & Grady, C. L. (1996). Face encoding and recognition in the human brain. *Proceedings of the National Academy of Sciences*, 93, 922-927.
- Heiss, W.-D., & Thiel, A. (2006). A proposed regional hierarchy in recovery of post-stroke aphasia. *Brain and Language*, 98, 118-123.
- Helm-Estabrooks, N. (1998). A "cognitive" approach to treatment of an aphasic patient. In N. Helm-Estabrooks & A. L. Holland (Eds.), *Approaches to the treatment of aphasia* (pp. 68-89). San Diego, CA: Singular.
- Helm-Estabrooks, N. (2001). Aphasia and nonlinguistic aspects of cognition. *Brain and Language*, 79, 8-9.
- Helm-Estabrooks, N. (2002). Cognition and aphasia: a discussion and a study. *Journal of Communication Disorders*, 35, 171-186.
- Helm-Estabrooks, N., & Albert, M. L. (1991). *Manual of aphasia therapy*. Austin, TX: Pro-Ed.
- Helm-Estabrooks, N., Bayles, K., Ramage, A. E., & Bryant, S. (1995). Relationship between cognitive performance and aphasia severity, age, and education: females versus males. *Brain and Language*, 51, 139-141.
- Helm-Estabrooks, N., Connor, L. T., & Albert, M. L. (2000). Treating attention to improve auditory comprehension in aphasia. *Brain and Language*, 74, 469-472.
- Hinckley, J. J., & Carr, T. H. (2001). Differential contributions of cognitive abilities to success in skill-based versus context-based aphasia treatment. *Brain and Language*, 79, 3-6.

- Hjelmquist, E. K. (1989). Concept formation in non-verbal categorization tasks in brain-damaged patients with and without aphasia. *Scandinavian Journal of Psychology*, 30, 243-254.
- Hockey, A., & Geffen, G. M. (2004). The concurrent validity and test-retest reliability of a visuospatial working memory task. *Intelligence*, 32, 591-605.
- Honey, G. D., & Fletcher, P. C. (2006). Investigating principles of human brain function underlying working memory: What insights from schizophrenia? . *Neuroscience*, 139, 59-71.
- Hulme, C., Roodenrys, S., & Mercer, R. (1995). The role of long-term memory mechanisms in memory span. *British Journal of Psychology* 86, 527-536.
- Huynh, H., & Feldt, L. S. (1976). Estimation of the Box correction for degrees of freedom from sample data in randomized block and split-plot designs. *Journal of Education Statistics*, 1, 69-82.
- Jarrold, C., & Towse, J. N. (2006). Individual differences in working memory. *Neuroscience*, 139, 39-50.
- Jones-Gotman, M., & Milner, B. (1977). Design fluency: the investigation of nonsense drawings after focal cortical lesions. *Neuropsychologia*, 15, 653-674.
- Jusczyk, P. W. (1997). *The discovery of spoken language (pp. 197-231)*. Cambridge, MA: MIT Press.
- Just, M. A., & Carpenter, P. A. (1992). A capacity theory of comprehension: Individual differences in working memory. *Psychological Review*, 99(1), 122-149.

- Kahneman, D. (1973). *Attention and effort*. Englewood Cliffs, NJ: Prentice-Hall.
- Kane, M. J., Bleckley, M. K., Conway, A. R. A., & Engle, R. W. (2001). A controlled-attention view of working memory capacity. *Journal of Experimental Psychology: General*, 130(2), 169-183.
- Kane, M. J., & Engle, R. W. (2002). The role of prefrontal cortex in working memory capacity, executive attention, and general fluid intelligence: An individual-differences perspective. *Psychonomic Bulletin & Review*, 9(4), 637-671.
- Kane, M. J., Hambrick, D. Z., Tuholski, S. W., Wilhelm, O., Payne, T. W., & Engle, R. W. (2004). The generality of working memory capacity: A latent-variable approach to verbal and visuospatial memory span and reasoning. *Journal of Experimental Psychology: General*, 133(2), 189-217.
- Kaplan, E., Gallagher, R. E., & Glosser, G. (1998). Aphasia-related disorders. In M. T. Sarno (Ed.), *Acquired aphasia* (Third ed., pp. 309-339). San Diego, CA: Academic Press.
- Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *Boston Naming Test*. Philadelphia: Lea & Febiger.
- Karbe, H., Thiel, A., Weber-Luxenburger, G., Herholz, K., Kessler, J., & Heiss, W.-D. (1998). Brain plasticity in poststroke aphasia: What is the contribution of the right hemisphere. *Brain and Language*, 64, 215-230.
- Keil, K. (2003). Executive function and aphasia. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 63(8-B), 3921.
- Keil, K., & Kaszniak, A. W. (2002). Examining executive function in individuals with brain injury: a review. *Aphasiology*, 16(3), 305-355.



- Kelley, W. M., Miezin, F. M., McDermott, K. B., Buckner, R. L., Raichle, M. E., Cohen, N. J., et al. (1998). Hemispheric specialization in human dorsal frontal cortex and medial temporal lobe for verbal and nonverbal memory encoding. *Neuron*, 20, 927-936.
- Kertesz, A. (1982). *Western Aphasia Battery*. Austin, TX: Pro-Ed.
- Kertesz, A., & McCabe, P. (1975). Intelligence and aphasia: performance of aphasics on Raven's Coloured Progressive Matrices (RCPM). *Brain and Language*, 2, 387-395.
- Kimberg, D. Y., D'Esposito, M., & Farah, M. J. (1997). Cognitive functions in the prefrontal cortex: Working memory and executive control. *Current Directions in Psychological Science*, 6, 185-192.
- Kiss, I., Pazderka-Robinson, H., & Floden, D. (2001). Event-Related Brain Potentials and central executive function: Further evidence for Baddeley's model. *Journal of Psychophysiology*, 15, 1-13.
- Kolk, H. H. J., & van Grunsven, M. F. (1985). Agrammatism as a variable phenomenon. *Cognitive Neuropsychology*, 2(4), 347-384.
- Konishi, S., Hayashi, T., Uchida, I., Kikyo, H., Takahashi, E., & Miyashita, Y. (2002). Hemispheric asymmetry in human lateral prefrontal cortex during cognitive set shifting. *Proceedings of the National Academy of Sciences*, 99(11), 7803-7808.
- Kucera, F., & Francis, W. (1967). *Computational analysis of present-day American English*. Providence, RI: Brown University Press.

- Lang, C. J. (1989). Continuous figure recognition in dementia and unilateral cerebral damage. *Neuropsychologia*, 25(5), 619-628.
- LaPointe, L. B., & Engle, R. W. (1990). Simple and complex word spans as measures of working memory capacity. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 16, 1118-1133.
- LaPointe, L. L., & Erickson, R. J. (1991). Auditory vigilance during divided task attention in aphasic individuals. *Aphasiology*, 5, 511-520.
- Larrabee, G. J. (1986). Another look at VIQ-PIQ scores and unilateral brain damage. *International Journal of Neuroscience*, 29, 141-148.
- Lashley, K. S. (1929). *Brain mechanisms and intelligence*. Chicago: University of Chicago Press.
- Laures, J. S. (2005). Reaction time and accuracy in individuals with aphasia during auditory vigilance tasks. *Brain and Language*, 95(2), 353-357.
- Laures, J. S., Odell, K., & Coe, C. L. (2003). Arousal and auditory vigilance in individuals with aphasia during a linguistic and nonlinguistic task. *Aphasiology*, 17(12), 1133-1152.
- Leker, R. R., & Shohami, E. (2002). Cerebral ischemia and trauma -- different etiologies yet similar mechanisms: neuroprotective opportunities. *Brain Research Reviews*, 39, 55-73.
- Levelt, W. J. M. (1999). Models of word production. *Trends in Cognitive Sciences*, 3(6), 223-232.
- Levelt, W. J. M., Roelofs, A., & Meyer, A. S. (1999). A theory of lexical access in speech production. *Behavioral and Brain Sciences*, 22, 1-75.

- Levin, H. S., Hanten, G., Chang, C.-C., Zhang, L., Schachar, R., Ewing-Cobbs, L., et al. (2002). Working memory after traumatic brain injury in children. *Annals of Neurology*, 52, 82-88.
- Lezak, M. D. (1993). Newer contributions to the neuropsychological assessment of executive functions. *Journal of Head Trauma Rehabilitation*, 8(1), 24-31.
- Lincoln, N. B., Majid, M. J., & Weyman, N. (2003). Cognitive rehabilitation for attention deficits following stroke (Cochrane Review). In *The Cochrane Library, Issue 4*. Chichester, UK: John Wiley & Sons, Ltd.
- Linebarger, M., McCall, D., Virata, T., & Berndt, R. S. (2007). Widening the temporal window: Processing support in the treatment of aphasic language production. *Brain and Language*, 100, 53-68.
- Logie, R. H., Della Sala, S., Cocchini, G., & Baddeley, A. D. (2004). Is there a specific executive capacity for dual task coordination? Evidence from Alzheimer's Disease. *Neuropsychology*, 18(3), 504-513.
- Luce, P. A., & Pisoni, D. (1998). Recognizing spoken words-- The neighborhood activation model. . *Ear and Hearing*, 19, 1-36.
- Luria, A. R. (1961). *The role of speech in the regulation of normal and abnormal behavior*. Oxford: Pergamon Press.
- Luria, A. R. (1966). *Higher cortical functions in man*. London: Tavistock.
- Luria, A. R. (1973). *The working brain*. New York: Penguin Books.
- Luzzatti, C., Raggi, R., Zonca, G., Pistarini, C., Contardi, A., & Pinna, G.-D. (2002). Verb-noun double dissociation in aphasic lexical impairments: the role of word frequency and imageability. *Brain and Language*, 81, 432-444.

- Maas, E., Barlow, J., Robin, D., & Shapiro, L. (2002). Treatment of sound errors in aphasia and apraxia of speech: Effects of phonological complexity. *Aphasiology*, 16(45/46), 609-622.
- MacDonald, M. C., & Christiansen, M. H. (2002). Reassessing working memory: Comment on Just and Carpenter (1992) and Waters and Caplan (1996). *Psychological Review*, 109(1), 35-54.
- Mainela-Arnold, E., & Evans, J. (2004, June 3-5, 2004). *The impact of lexical representations on verbal working memory capacity in children with specific language impairment*. Paper presented at the 25th Annual Symposium of Research in Child Language Disorders, Madison, WI.
- Mainela-Arnold, E., & Evans, J. (2005). Beyond capacity limitations: determinants of word recall performance on verbal working memory span tasks in children with SLI. *Journal of Speech, Language, and Hearing Research*, 48, 897-909.
- Mainela-Arnold, E., Evans, J., & Coady, J. (2005). *The nature of lexical representations in children with SLI: Evidence from a frequency adjusted forward gating task*. Paper presented at the Symposium on Research in Child Language Disorders, Madison, WI.
- Martin, N., Saffran, E. M., & Dell, G. S. (1996). Recovery in deep dysphasia: Evidence for a relation between auditory-verbal STM capacity and lexical errors in repetition. *Brain and Language*, 52, 83-113.
- Martin, R. C. (1995). Working memory doesn't work: A critique of Miyake et al.'s capacity theory of aphasic comprehension deficits. *Cognitive Neuropsychology*, 12(6), 623-636.

- Martin, R. C., Breedin, S. D., & Damian, M. F. (1999). The relation of phoneme discrimination, lexical access, and short-term memory: A case study and interactive activation account. *Brain and Language*, 70, 437-482.
- Martin, R. C., & Feher, E. (1990). The consequences of reduced memory span for the comprehension of semantic versus syntactic information. *Brain and Language*, 38, 1-20.
- Martin, R. C., & Freedman, M. L. (2001). Short-term retention of lexical-semantic representations for speech production. *Memory*, 9(4/5/6), 261-280.
- Martin, R. C., Lesch, M. F., & Bartha, M. C. (1999). Independence of input and output phonology in word processing and short-term memory. *Journal of Memory and Language*, 41, 3-29.
- Martinkauppi, S., Rama, P., Aronen, H. J., Korvenoja, A., & Carlson, S. (2000). Working memory of auditory localization. *Cerebral Cortex*, 10, 889-898.
- Marton, K., & Schwartz, R. G. (2003). Working memory capacity and language processes in children with Specific Language Impairment. *Journal of Speech, Language, and Hearing Research*, 46, 1138-1153.
- Mayer, J. F., & Murray, L. L. (2002). Approaches to the treatment of alexia in chronic aphasia. *Aphasiology*, 16(7), 727-743.
- McNeil, M. R., Hula, W. D., Matthews, C. T., & Doyle, P. J. (2004). Resource theory and aphasia: A fugacious theoretical dismissal. *Aphasiology*, 18(9), 836-839.
- McNeil, M. R., Hula, W. D., Matthews, C. T., Doyle, P. J., & Fossett, T. R. D. (2004). Auditory comprehension and visual-manual tracking dual-task

- performance in aphasia: Preliminary findings. *Brain and Language*, 91(1), 31-32.
- McNeil, M. R., Odell, K., & Tseng, C.-H. (1991). Toward the integration of resource allocation into a general theory of aphasia. *Clinical Aphasiology*, 20, 21-39.
- Meegan, D. V., Pure-Stephenson, R., Honsberger, M. J., & Topan, M. (2003). Task analysis complements neuroimaging: an example from working memory research. *NeuroImage*, 21, 1026-1036.
- Mitchell, J. P., Macrae, N., & Gillchrist, I. D. (2002). Working memory and the suppression of reflexive saccades. *Journal of Cognitive Neuroscience*, 14(1), 95-103.
- Miyake, A., Carpenter, P. A., & Just, M. A. (1994). A capacity approach to syntactic comprehension disorders: Making normal adults perform like aphasic patients. *Cognitive Neuropsychology*, 11, 671-717.
- Miyake, A., Carpenter, P. A., & Just, M. A. (1995). Reduced resources and specific impairments in normal and aphasic sentence comprehension. *Cognitive Neuropsychology*, 12(6), 651-679.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: a latent variable analysis. *Cognitive Psychology*, 41, 49-100.
- Moineau, S., Dronkers, N. F., & Bates, E. (2005). Exploring the processing continuum of single-word comprehension in aphasia. *Journal of Speech, Language, and Hearing Research*, 48, 884-896.

- Monetta, L., & Pell, M. D. (2006). Effects of verbal working memory deficits on metaphor comprehension in patients with Parkinson's disease. *Brain and Language, in press*.
- Morrisette, M. L., & Gierut, J. A. (2002). Lexical organization and phonological change in treatment. *Journal of Speech, Language, and Hearing Disorders, 45*, 143-159.
- Morrison, C. M., Ellis, A. W., & Quinlan, P. T. (1992). Age of acquisition, not word frequency, affects object naming, not object recognition. *Memory & Cognition, 20*(6), 705-714.
- Mottaghy, F. M. (2006). Interfering with working memory in humans. *Neuroscience, 139*, 85-90.
- Mueller, S. T., Seymour, T. L., Kieras, D. E., & Meyer, D. E. (2003). Theoretical implications of articulatory duration, phonological similarity, and phonological complexity in verbal working memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 29*(6), 1353-1380.
- Murray, L. L. (1999). Attention and aphasia: theory, research, and clinical implications. *Aphasiology, 13*(2), 91-111.
- Murray, L. L. (2000). The effects of varying attentional demands on the word retrieval skills of adults with aphasia, right hemisphere brain damage, or no brain damage. *Brain and Language, 72*, 40-72.
- Murray, L. L. (2003, November 2003). *Cognitive treatments for aphasia: should we and can we help attention and working memory?* Paper presented at the Academy of Neurologic Communication Disorders and Sciences, Chicago, IL.

- Murray, L. L., Baldwin, R. J., & Karcher, L. (2002). *Treating attention in mild aphasia: evaluation of Attention Process Training-II*. Paper presented at the American Speech, Language, and Hearing Association, Atlanta, GA.
- Murray, L. L., Holland, A. L., & Beeson, P. M. (1997a). Auditory processing in individuals with mild aphasia: a study of resource allocation. *Journal of Speech, Language, and Hearing Research*, 40, 792-808.
- Murray, L. L., Holland, A. L., & Beeson, P. M. (1997b). Grammaticality judgments of mildly aphasic individuals under dual-task conditions *Aphasiology*, 11, 993-1016.
- Murray, L. L., & Kean, J. (2004). Resource theory and aphasia: time to abandon or time to revise? *Aphasiology*, 18(9), 830-835.
- Murray, L. L., & Ramage, A. E. (2000a, May 2000). *Assessing the executive function abilities of adults with aphasia: why, how, and even some data*. Paper presented at the Clinical Aphasiology Conference, Waikoloa Beach, HA.
- Murray, L. L., & Ramage, A. E. (2000b). Assessing the executive function abilities of adults with neurogenic communication disorders. *Seminars in Speech and Language*, 21(1), 153-168.
- Nation, K., Adams, J. W., Bowyer-Crane, C. A., & Snowling, M. J. (1999). Working memory deficits in poor comprehenders reflect underlying language impairments. *Journal of Experimental Child Psychology*, 73, 139-158.
- Navon, D. (2004). Aphasia, resources, and cross-talk: Do we now have a soup mix? *Aphasiology*, 18(9), 840-843.



- Neumar, R. W. (2000). Molecular mechanisms of ischemic neuronal injury. *Annals of Emergency Medicine*, 36(5), 483-506.
- Nicholas, M. L. (1999). Category selection in patients with nonfluent aphasia: Implications for use of a picture-based alternative communication system. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 59(7-B), 3392.
- Nickels, L., & Howard, D. (1995). Aphasic naming: What matters? *Neuropsychologia*, 33(10), 1281-1303.
- Noppeney, U., & Wallesch, C.-W. (2000). Language and cognition: Kurt Goldstein's theory of semantics. *Brain and Cognition*, 44, 367-386.
- Nusbaum, H., Pisoni, D., & Davis. (1984). Hoosier Mental Lexicon. from <http://128.252.27.56/neighborhood/home.asp>
- Nusbaum, H., & Small, S. (2001). Perceptual learning of spoken language: cognitive mechanisms and implications for aphasia recovery. *Brain and Language*, 79, 7-8.
- Nystrom, L. E., Braver, T. S., Sabb, F. W., Delgado, M. R., Noll, D. C., & Cohen, J. D. (2000). Working memory for letters, shapes, and locations: fMRI evidence against stimulus-based regional organization in human prefrontal cortex. *NeuroImage*, 11, 424-446.
- Osaka, M., Osaka, N., Kondo, H., Morishita, M., Fukuyama, H., Aso, T., et al. (2003). The neural basis of individual differences in working memory capacity: An fMRI study. *NeuroImage*, 18, 789-797.

- Ostergaard, A. L., & Meudell, P. R. (1984). Immediate memory span, recognition memory for subspan series of words, and serial position effects in recognition memory for supraspan series of verbal and nonverbal items in Broca's and Wernicke's aphasia. *Brain and Language*, 22, 1-13.
- Papagno, C., & Basso, A. (1996). Perseveration in two aphasic patients. *Cortex*, 32, 67-82.
- Parmenter, B. A., Shucard, J. L., Benedict, R. H. B., & Shucard, D. W. (2006). Working memory deficits in multiple sclerosis: Comparison between the n-back task and the Paced Auditory Serial Addition Test. *Journal of the International Neuropsychological Society*, 12, 677-687.
- Pashek, G. V., & Tompkins, C. A. (2002). Context and word class influences on lexical retrieval in aphasia. *Aphasiology*, 16(3), 261-286.
- Perlstein, W. M., Cole, M. A., Demery, J. A., Seignourel, P. J., Dixit, N. K., Larson, M. J., et al. (2004). Parametric manipulation of working memory load in traumatic brain injury: behavioral and neural correlates. *Journal of the International Neuropsychological Society*, 10, 724-741.
- Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal and temporal-lobe lesion in man. *Neuropsychologia*, 20, 249-262.
- Petry, M. C., Crosson, B., Gonzalez-Rothi, L. J., Bauer, R. M., & Schauer, C. A. (1994). Selective attention and aphasia in adults: preliminary findings. *Neuropsychologia*, 32(11), 1397-1408.

- Pexman, P. M., Lupker, S. J., & Hino, Y. (2002). The impact of feedback semantics in visual word recognition: Number-of-features effects in lexical decision and naming tasks. *Psychonomic Bulletin and Review*, 9(3), 542-549.
- Pollak, S., & Friesen, W. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, 112(3), 323-338.
- Posner, M. I. (1994). Attention: The mechanisms of consciousness. *Proceedings of the National Academy of Sciences*, 91, 7398-7403.
- Postle, B. R. (2006). Working memory as an emergent property of the mind and brain. *Neuroscience*, 139, 23-38.
- Postle, B. R., D'Esposito, M., & Corkin, S. (2005). Effects of verbal and nonverbal interference on spatial and object visual working memory *Memory and Cognition*, 33, 203-212.
- Purdy, M. (2002). Executive function ability in persons with aphasia. *Aphasiology*, 16(4/5/6), 549-557.
- Ramachandran, V. S. (2005). Plasticity and functional recovery in neurology. *Clinical Medicine*, 5(4), 368-373.
- Ramsberger, G. (1994). Functional perspective for assessment and rehabilitation of persons with severe aphasia. *Seminars in Speech and Language*, 15(1), 1-16.
- Ramsberger, G. (2005). Achieving conversational success in aphasia by focusing on non-linguistic cognitive skills: A potentially promising new approach. *Aphasiology*, 19(10/11), 1066-1073.

- Ranganath, C. (2006). Working memory for visual objects: Complementary roles of inferior temporal, medial temporal, and prefrontal cortex. *Neuroscience*, 139, 277-289.
- Raven, J., Raven, J. C., & Court, J. H. (1998). *Coloured Progressive Matrices*. Oxford: Oxford Psychologists Press.
- Raven, J. C., Court, J. H., & Raven, J. (1985). Manual for Raven's Progressive Matrices and Vocabulary Scales. In. London: H. K. Lewis.
- Raven, J. C., Court, J. H., & Raven, J. (1999). *Standard Progressive Matrices*. Oxford: Oxford Psychologists Press.
- Raymer, A. M., & Gonzalez-Rothi, L. J. (2001). Cognitive approaches to impairments of word comprehension and production. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorders* (pp. 524-550). Philadelphia: Lippincott, Williams, & Wilkins.
- Repovs, G., & Baddeley, A. D. (2006). The multi-component model of working memory: explorations in experimental cognitive psychology. *Neuroscience*, 139, 5-21.
- Roberts, R., & Gibson, E. (2002). Individual differences in sentence memory. *Journal of Psycholinguistic Research*, 31(6), 573-598.
- Robin, D. A., & Rizzo, M. (1989). The effect of focal cerebral lesions on intramodal and cross-modal orienting of attention. *Clinical Aphasiology*, 18, 61-74.
- Rochon, E., Waters, G. S., & Caplan, D. (1994). Sentence comprehension in patients with Alzheimers disease. *Brain and Language*, 46, 329-349.

- Rochon, E., Waters, G. S., & Caplan, D. (2000). The relationship between measures of working memory and sentence comprehension in patients with Alzheimer's Disease. *Journal of Speech, Language, and Hearing Research, 43*, 395-413.
- Roodenrys, S., Hulme, C., Lethbridge, A., Hinton, M., & Nimmo, L. (2002). Word frequency and phonological neighborhood effects on verbal short-term memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 28*, 1019-1034.
- Rosch, E. (1975). Cognitive representations of semantic categories. *Journal of Experimental Psychology: General, 104*(3), 192-233.
- Salthouse, T. A. (1995). Differential age-related influences on memory for verbal-symbolic information and visual-spatial information. *Journal of Gerontology: Psychological Sciences, 50B*, 193-201.
- Salthouse, T. A. (2004). *Investigation of construct validity of executive functioning*. Paper presented at the International Neuropsychological Society Thirty-Second Annual Meeting.
- Salthouse, T. A., Atkinson, T. M., & Berish, D. E. (2003). Executive functioning as a potential mediator of age-related cognitive decline in normal adults. *Journal of Experimental Psychology: General, 132*(4), 566-594.
- Schuell, H. (1965). *The Minnesota test for differential diagnosis of aphasia*. Minneapolis: University of Minnesota Press.
- Shah, P., & Miyake, A. (1996). The separability of working memory resources for spatial thinking and language processing: An individual differences approach. *Journal of Experimental Psychology: General, 125*(1), 4-27.

- Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London, Series B*, 298, 199-209.
- Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain*, 114, 727-741.
- Shisler, R. J. (2005). Aphasia and auditory extinction: Preliminary evidence of binding. *Aphasiology*, 19(7), 633-650.
- Shuster, L. I. (2004). Resource theory and aphasia reconsidered: Why alternative theories can better guide our research. *Aphasiology*, 18(9), 811-854.
- Shuster, L. I., & Thompson, J. C. (2004). Resource theory: Here, there, everywhere. *Aphasiology*, 18(9), 850-854.
- Silkes, J. P., McNeil, M. R., & Drton, M. (2004). Simulation of aphasic naming performance in non-brain-damaged adults. *Journal of Speech, Language, and Hearing Research*, 47, 610-623.
- Simmons, M. R. (2001). The central executive and working memory: A dual-task investigation of the n-back task. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 61(8-B), 4442.
- Smith, E. E., Geva, A., Jonides, J., Miller, A., Reuter-Lorenz, P., & Koeppel, R. A. (2001). The neural basis of task-switching in working memory: Effects of performance and aging. *Proceedings of the National Academy of Sciences*, 98(4), 2095-2100.
- Smith, E. E., & Jonides, J. (1997). Working memory: A view from neuroimaging. *Cognitive Psychology*, 33, 5-42.

- Smith, E. E., & Jonides, J. (1999). Storage and executive processes in the frontal lobes. *Science*, 283, 1657-1661.
- Smith, E. E., Jonides, J., Marshuetz, C., & Koeppel, R. A. (1998). Components of verbal working memory: Evidence from neuroimaging. *Proceedings of the National Academy of Sciences, U.S.A.*, 95, 876-882.
- Sohlberg, M. M., Johnson, L., Paule, L., Raskin, S. A., & Mateer, C. A. (1994). *Attention Process Training II: A program to address attentional deficits for persons with mild cognitive dysfunction*. Puyallup, WA: Association for Neuropsychological Research and Development.
- Spree, O., & Strauss, E. (1998). *A compendium of neuropsychological tests* (Second ed.). New York: Oxford University Press.
- Sturm, W., & Willmes, K. (1991). Efficacy of a reaction training on various attentional and cognitive functions in stroke patients. *Neuropsychological Rehabilitation*, 1, 259-280.
- Swanson, H. L., & Alexander, J. E. (1997). Cognitive processes as predictors of word recognition and reading comprehension in learning-disabled and skilled readers: Revisiting the specificity hypothesis. *Journal of Educational Psychology*, 89(1), 128-158.
- Sylvester, C.-Y. C., Wager, T. D., Lacey, S. C., Hernandez, L., Nichols, T. E., Smith, E. E., et al. (2003). Switching attention and resolving interference: fMRI measures of executive functions. *Neuropsychologia*, 41, 357-370.

- Tompkins, C. A., Bloise, C. G. R., Timko, M. L., & Baumgaertner, A. (1994). Working memory and inference revision in brain-damaged and normally aging adults. *Journal of Speech and Hearing Research*, 37, 896-912.
- Tseng, C.-H., McNeil, M. R., & Milenkovic, P. (1993). An investigation of attention allocation deficits in aphasia. *Brain and Language*, 45, 276-296.
- Valler, G., Corno, M., & Basso, A. (1992). Auditory and visual verbal short-term memory in aphasia. *Cortex*, 28, 383-389.
- Van Harskamp, F., & Visch-Brink, E. G. (1991). Goal recognition in aphasia therapy. *Aphasiology*, 5(6), 529-539.
- Van Mourik, M., Verschaeve, M., Boon, P., Paquier, P., & Van Harskamp, F. (1992). Cognition in global aphasia: indicators for therapy. *Aphasiology*, 6(5), 491-499.
- Verhaeghen, P., & Basak, C. (2005). Ageing and switching of the focus of attention in working memory: Results from a modified *N*-Back task. *The Quarterly Journal of Experimental Psychology*, 58A(1), 134-154.
- Vilkki, J. (1988). Problem solving deficits after focal cerebral lesions. *Cortex*, 24, 199-127.
- Vitevitch, M. S., & Luce, P. A. (2005). Increases in phonotactic probability facilitate spoken nonword repetition. *Journal of Memory and Language*, 52, 193-204.
- Waters, G. S., & Caplan, D. (1996). The measurement of verbal working memory capacity and its relation to reading comprehension. *Quarterly Journal of Experimental Psychology*, 49, 51-79.



- Waters, G. S., & Caplan, D. (2003). The reliability and stability of verbal working memory measures. *Behavior Research Methods, Instruments, & Computers*, 35(4), 550-564.
- Watter, S., Geffen, G. M., & Geffen, L. B. (2001). The n-back as a dual-task: P300 morphology under divided attention. *Psychophysiology*, 38, 998-1003.
- Wayland, S., & Taplin, J. E. (1982). Nonverbal categorization in fluent and nonfluent anomic aphasics. *Brain and Language*, 16, 87-108.
- Wayland, S., & Taplin, J. E. (1985). Feature-processing deficits following brain injury: II. Classification learning, categorical decision making, and feature production. *Brain and Cognition*, 4, 356-376.
- Ween, J. E., Verfaellie, M., & Alexander, M. P. (1996). Verbal memory function in mild aphasia. *Neurology*, 47(3), 795-801.
- Wepman, J. (1972). Aphasia therapy: A new look. *Journal of Speech and Hearing Disorders*, 37, 203-214.
- Williams, S. E. (1983). Factors influencing naming performance in aphasia: A review of the literature. *Journal of Communication Disorders*, 16, 357-372.
- Wright, H. H., Newhoff, M., Downey, R. A., & Austermann, S. (2003). *Additional data on working memory in aphasia*. Paper presented at the International Neuropsychological Society Annual Conference, Honolulu, Hawaii.
- Wright, H. H., & Shisler, R. J. (2005). Working memory in aphasia: theory, measures, and clinical implications. *American Journal of Speech-Language Pathology*, 14, 107-118.

Table 1. *Demographic Characteristics of the Participants With Aphasia (LHD) and Healthy Controls (NBD).*

	<i>LHD</i>	<i>NBD</i>
Gender ratio (F:M)	4:11	8:4
Age	54.6 (SD = 14.6)	52.8 (SD = 13.3)
Education	15.3 (SD = 3.0)	15.2 (SD = 2.4)
RCPM (TONI-III) %ile	36.5 (SD = 31.7)	74 (SD = 29.8)

Table 2. *Clinical Characteristics of the Participants With Aphasia*

<i>Subject</i>	<i>Age</i>	<i>TPO (mos.)</i>	<i>RCPM (or TONI-III) %ile rank</i>	<i>Aphasia Type (fluency)</i>	<i>WAB AQ</i>	<i>Years of Education</i>
S1	37	90	5	nonfluent	31.6	12
S2	60	5	0	fluent	70.6	16
S3	52	66	70	nonfluent	82.8	16
S4	47	18	95	nonfluent	57.9	16
S5	64	67	40*	fluent	95.4	12
S6	25	20	5	fluent	83.8	15
S7	72	30	45	fluent	93.4	12
S8	75	216	5	nonfluent	16.4	15
S9	68	132	55*	fluent	95.8	18
S10	52	38	55*	nonfluent	70.6	12
S11	47	12	5*	fluent	74.4	11
S12	57	264	DNT	nonfluent	DNT	20
S13	56	123	58*	nonfluent	44.9	16
S14	55	10	74*	fluent	82.4	20
S15	53	84	16*	nonfluent	79	18

*Note.* TPO, time post-onset; RCPM, Raven's Colored Progressive Matrices; TONI-III, Test of Nonverbal Intelligence, 3<sup>rd</sup> edition; WAB AQ, Western Aphasia Battery aphasia quotient; DNT, did not test; \* denotes patients who were administered the TONI-III.

Table 3. *Stimulus Characteristics (Mean, SD, Range)*

	<i>High Frequency Stimuli</i>	<i>Low Frequency Stimuli</i>
Kucera-Frances Frequency	240.3 (244.9, 75-1207) <sup>a</sup>	8.9 (9.46, 1-45)
Familiarity	594.6 (31.4, 541-645)	511.3 (58.5, 380-595)
Age of Acquisition	206.58 (39.0, 150-278)	255.0 (68.5, 186-386)
Concreteness	592.0 (36.4, 487-622)	584.8 (33.9, 513-636)
Imagery	598.8 (34.7, 525-639)	578.4 (40.2, 488-635)
Number of letters	4.0 (.83, 3-6)	3.6 (.72, 3-5)
Number of phonemes	3.0 (.72, 2-5)	3.0 (.36, 2-4)

- a. The large variation in high-frequency stimuli is due to including the stimulus word “man,” with a frequency count of 1207. The range of high frequency stimulus counts without this outlier was 75 (“dog”) to 591 (“house”).

Table 4. *Linguistic Stimuli Grouped According to Frequency Count*

<i>High Frequency</i>	<i>Low Frequency</i>
Dog	Frog
Boy	Bat
Key	Leaf
Girl	Pig
Sun	Spoon
Ball	Pail
Car	Eggs
Man	Pan
House	Ant
Room	Mop
Hair	Fork
Judge	Dice
Road	Cup
Ship	Goose
Watch	Goat
Food	Web
Bed	Mat
Teeth	Jar
Pool	Wheat
Door	Bun
Church	Fin
Fire	Kite
Hand	Bib
Plane	Rat

Table 5. *Mean Pr Values, Collapsed Across Groups, at Three Language Levels (High- and Low-Frequency Objects, Faces) and Three WM Load Levels (n= 0, 1, 2).*

<i>Language Load (stimuli)</i>	<i>WM Load (n)</i>	<i>Mean Pr</i>	<i>Std. Error</i>
High-frequency objects	n=0	.98	.01
	n=1	.93	.02
	n=2	.76	.02
Low frequency objects	n=0	.97	.02
	n=1	.96	.01
	n=2	.73	.03
Faces	n=0	.95	.02
	n=1	.85	.02
	n=2	.59	.04

Table 6. *Mean Pr Values for the LHD Group Only, at Three Language Levels (High- and Low-Frequency Objects, Faces) and Three WM Load Levels (n= 0, 1, 2).*

<i>Language Load (stimuli)</i>	<i>WM Load (n)</i>	<i>Mean Pr</i>	<i>Std. Error</i>
High-frequency objects	n=0	.96	.02
	n=1	.90	.03
	n=2	.63	.03
Low frequency objects	n=0	.94	.04
	n=1	.93	.02
	n=2	.58	.04
Faces	n=0	.90	.04
	n=1	.77	.04
	n=2	.44	.06

Table 7. LHD Group Correlations (*r*) Between Selected Variables.

	<i>AQ</i>	<i>RCPM</i>	<i>Name</i>	<i>Pr</i> <i>high</i>	<i>Pr</i> <i>low</i>	<i>Pr</i> <i>face</i>	<i>Pr 0</i>	<i>Pr 1</i>	<i>Pr 2</i>	<i>RT</i> <i>0</i>	<i>RT 1</i>	<i>RT 2</i>
<i>Age</i>	.04	.15	.12	-.5	-.42	-.58*	-.34	-.39	-.48	.08	.22	.21
<i>Educ</i>	.11	.39	.18	.07	.32	.17	-.38	-.21	.38	.57*	.26	.57*
<i>AQ</i>		.26	.92**	-.06	-.12	.53*	.22	.14	.15	-.11	-.11	-.13
<i>RCPM</i>			.32	.14	.14	.23	.06	.28	.15	.35	-.14	.61*
<i>Name</i>				.06	-.04	.62*	.18	.07	.24	-.08	-.09	-.05
<i>Pr</i>					.77**	.67**	.78**	.73**	.69**	-.4	-.43	-.22
<i>high</i>												
<i>Pr low</i>						.56*	.56*	.82**	.7**	-.12	-.42	-.08
<i>Pr face</i>							.72**	.68**	.75**	-.22	-.27	-.11
<i>Pr 0</i>								.82**	.39	-.39	-.34	-.41
<i>Pr 1</i>									.47	-.38	-.66*	-.24
<i>Pr 2</i>										-.17	-.16	.16
<i>RT 0</i>											.65**	.69**
<i>RT 1</i>												.48

Note: *Educ* = years of education; *AQ* = WAB Aphasia Quotient; *RCPM* = Raven's

Colored Progressive Matrices; *Name* = Picture naming task; *Pr high*, *Pr low*, *Pr face*

= *Pr* summed across WM load (0-, 1-, and 2-back) for high frequency, low frequency,

and face stimuli, respectively; *Pr 0*, *Pr 1*, *Pr 2* = *Pr* summed across language

conditions (high frequency, low frequency, faces) at the 0-, 1-, and 2-back level,

respectively; *RT 0*, *RT 1*, *RT 2* = RT summed across language conditions at the 0-, 1-,

and 2-back level, respectively; \* =  $p < .05$ , \*\* =  $p < .01$ .



Table 8. *NBD Group Correlations (r) Between Selected Variables.*

	<i>RCPM</i>	<i>Pr</i> <i>high</i>	<i>Pr</i> <i>low</i>	<i>Pr</i> <i>face</i>	<i>Pr 0</i>	<i>Pr 1</i>	<i>Pr 2</i>	<i>RT 0</i>	<i>RT 1</i>	<i>RT 2</i>
<i>Age</i>	.61	-.44	-.54	-.59*	-.46	-.38	.58*	.17	.08	.28
<i>Educ</i>	.8	.152	.24	.2	-.15	.04	.27	-.22	-.35	-.36
<i>RCPM</i>		-.323	-.45	-.09	-.39	-.39	-.18	.3	.22	.32
<i>Pr high</i>			.77**	.84**	.11	.92**	.85**	.00	-.1	-.55
<i>Pr low</i>				.71*	.18	.55**	.9**	.01	.05	-.43
<i>Pr face</i>					.12	.75**	.93**	.18	.16	-.31
<i>Pr 0</i>						-.23	.16	.27	.23	.05
<i>Pr 1</i>							.65*	-.1	-.18	-.55
<i>Pr 2</i>								.15	.15	-.35
<i>RT 0</i>									.85**	.69**
<i>RT 1</i>										.82**

Note: *Educ* = years of education; *RCPM* = Raven's Colored Progressive Matrices; *Pr high*, *Pr low*, *Pr face* = *Pr* summed across WM load (0-, 1-, and 2-back) for high frequency, low frequency, and face stimuli, respectively; *Pr 0*, *Pr 1*, *Pr 2* = *Pr* summed across language conditions (high frequency, low frequency, faces) at the 0-, 1-, and 2-back level, respectively; *RT 0*, *RT 1*, *RT 2* = RT summed across language conditions at the 0-, 1-, and 2-back level, respectively; \* =  $p < .05$ , \*\* =  $p < .01$ .

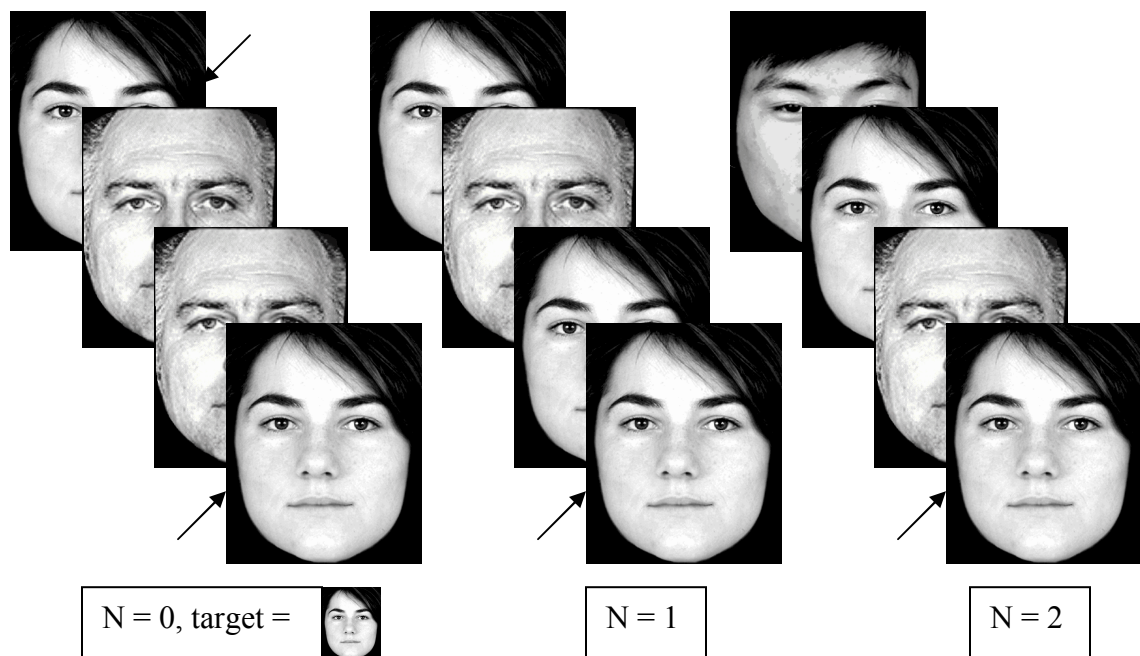


Figure 1. Schematic representation of the n-back tasks (face stimuli) at the 0, 1-, and 2-back levels. At the 0-back level, the participant's task is to decide whether each picture matches a pre-specified target. At the 1- and 2-back levels, the participant's task is to decide whether each picture matches one which occurred either one or two back in the sequence, respectively. Arrows denote hits.

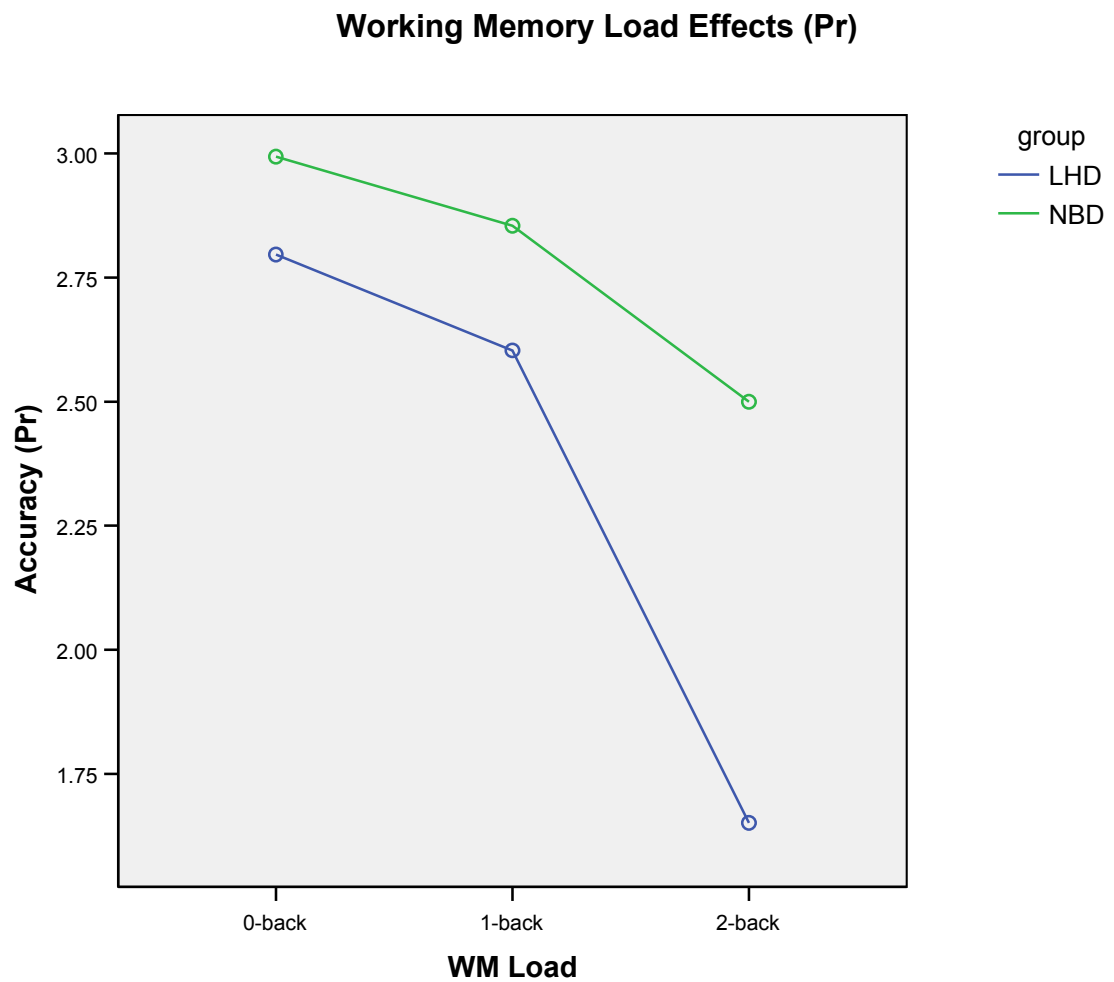


Figure 2. Group effects of WM load, collapsed (summed) across language stimulus conditions (high-frequency, low-frequency, and faces). The main effect of group, and interaction between group and WM load, are clearly visible.

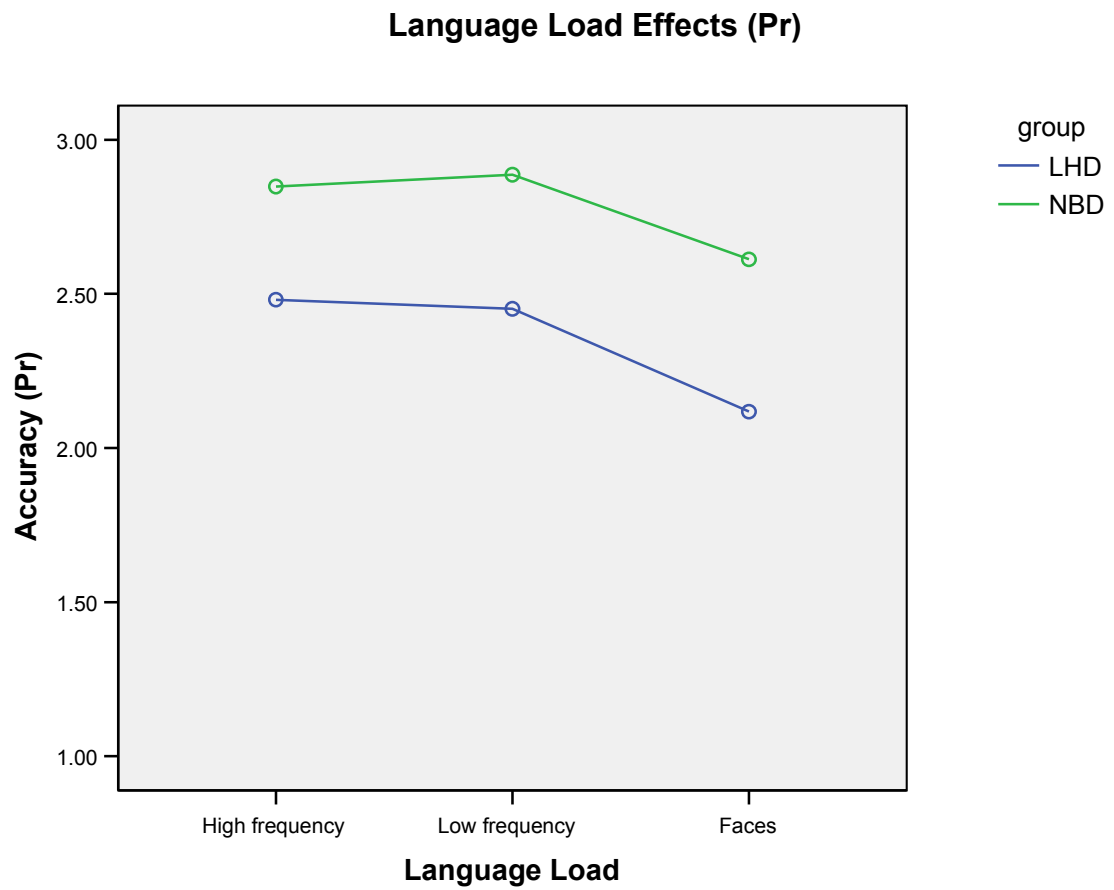


Figure 3. Group effects of language load (stimuli), collapsed (summed) across WM conditions (0-back, 1-back, 2-back). Note the parallel and relatively flat word frequency effect across groups.

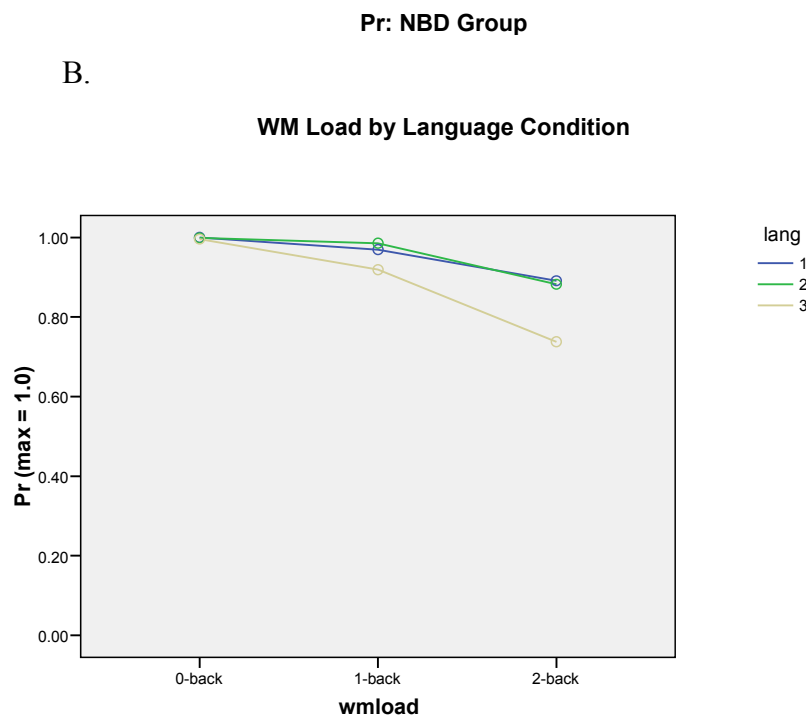
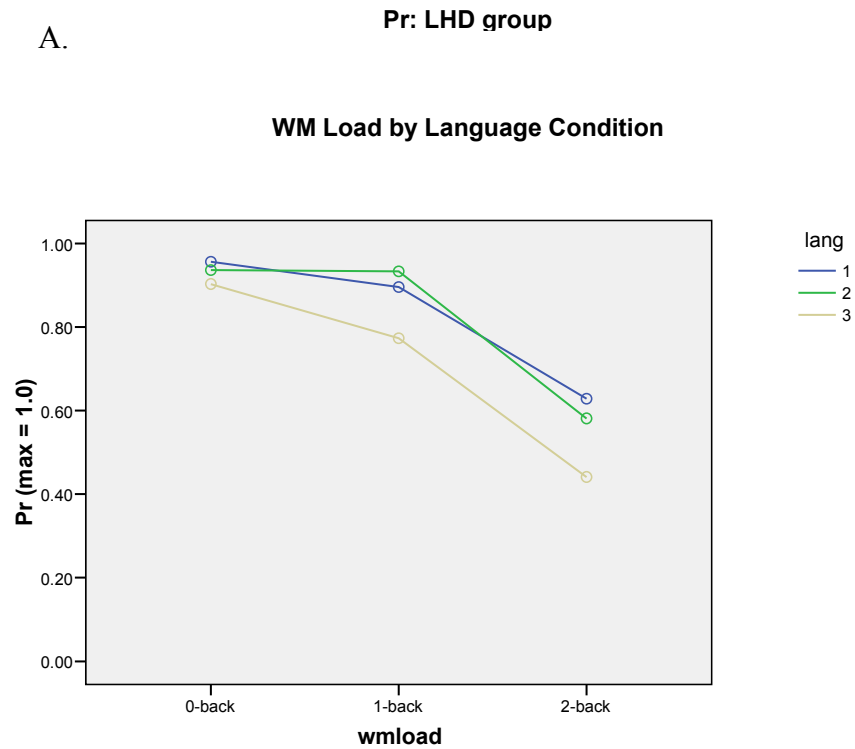


Figure 4. Pr for each group in relation to WM load (0-, 1-, and 2-back) and language condition (1=high frequency objects, 2=low frequency objects, 3=faces).

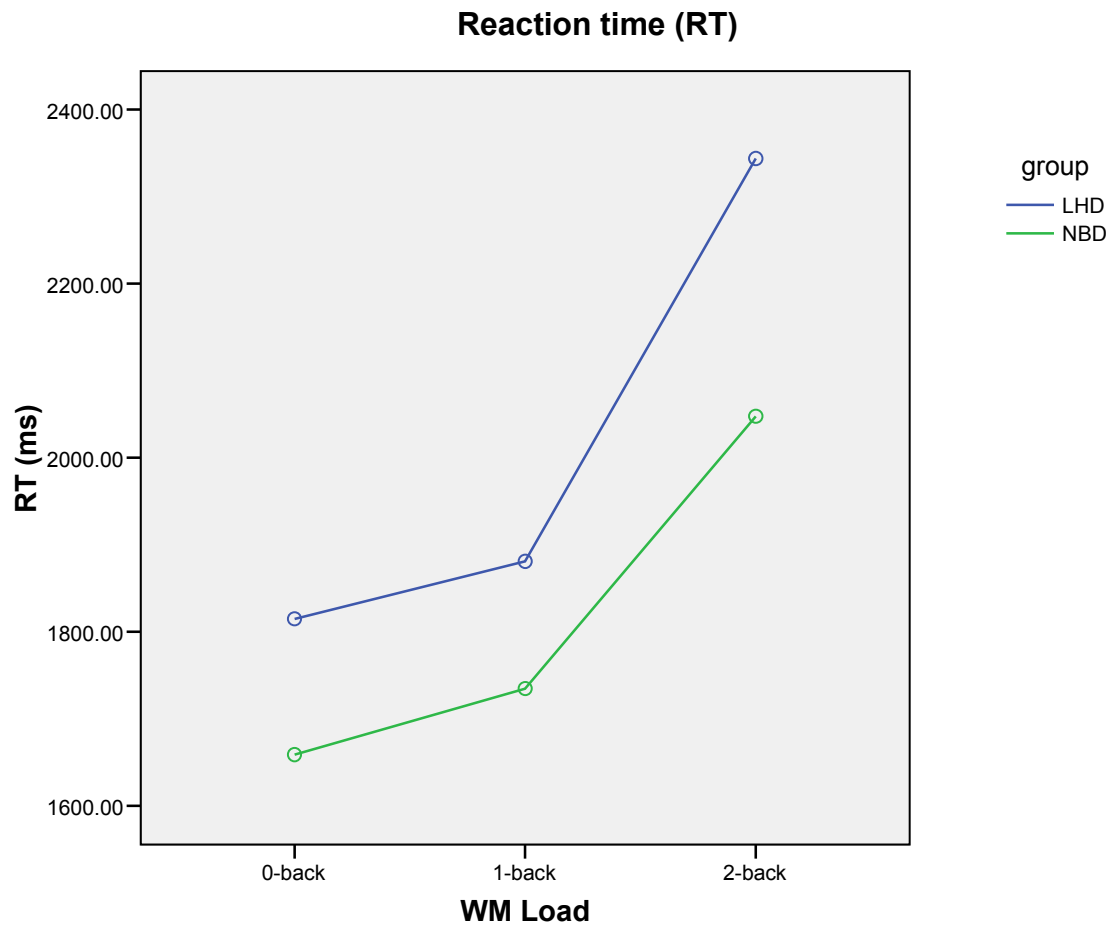
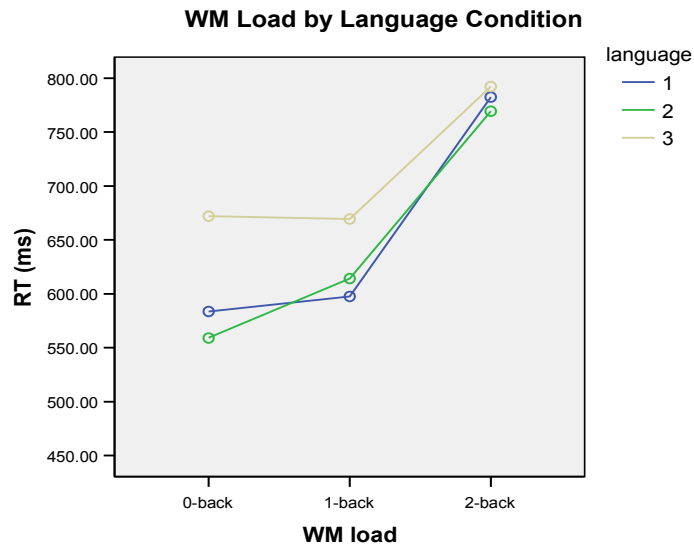
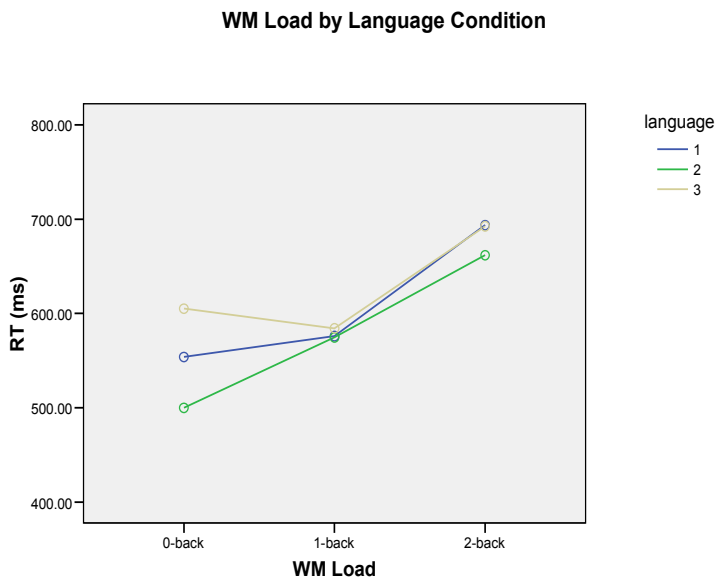


Figure 5. Reaction time data collapsed (summed) across language stimulus conditions. Note the parallel increases in RT across groups concurrent with increased WM load.

A. **Reaction time: LHD group**



B. **Reaction time: NBD group**



**Figure 6.** Reaction time (RT) for each group in relation to WM load (0-, 1-, and 2-back) and language condition (1=high frequency objects, 2=low frequency objects, 3=faces).

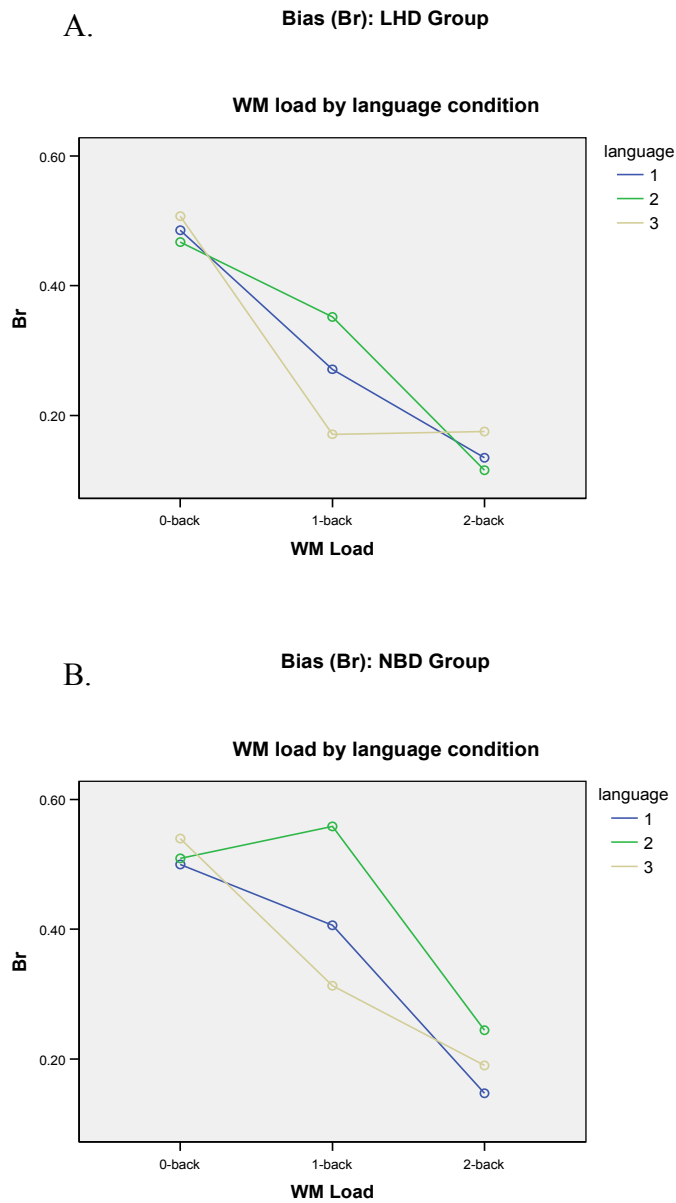


Figure 7. Bias (Br) for each group in relation to WM load (0-, 1-, and 2-back) and language condition (1 = high-frequency objects, 2 = low-frequency objects, 3 = faces). The higher the Br value, the more liberal the response (e.g., Br = 1: low number of misses, high number of false positives), and the lower the Br value, the more conservative the response (e.g., Br = 0: high number of misses, low number of false positives).



Study # 05-9791

INDIANA UNIVERSITY – BLOOMINGTON  
INFORMED CONSENT STATEMENT

**Working Memory and Aphasia**

You are invited to participate in a research study. The purpose of this study is to look at the link between language and memory in people with and without aphasia. This project may help healthcare professionals plan treatment programs for adults with left-hemisphere brain damage. Researchers at Indiana University are doing this study.

**INFORMATION**

**Procedures**

If you agree to be in this study, we will ask you to do these things:

- 1) Complete two language tests. These tests will involve listening, speaking, reading and writing. The tests will take about one and a half hours to complete.
- 2) Complete a test of thinking. This test will involve looking at pictures and patterns and will take about 15 minutes.
- 3) Complete two tasks on a computer. The tasks will involve paying attention and remembering. The tasks will take about one hour.

Thirty adults with aphasia, and thirty adults without brain damage, will be participating in this research.

**RISKS**

The main risk of being in this study is that you might get frustrated. This is just like in regular rehabilitation or therapy sessions. The researcher will do his or her best to make sure this happens as little as possible. The researcher will stop the testing session any time you ask, if you get tired or want to stop for any other reason.

**BENEFITS**

A benefit to being in the study is that you may learn information about what kinds of language or thinking tasks are easy and hard for you. Plus, this study will teach us more about how help other people with aphasia.

Subject's initials: \_\_\_\_\_

## **CONFIDENTIALITY**

The records of this research will be kept private in a locked office. Only the researcher will have access to these records. You will not be identified in any reports.

## **COMPENSATION**

You will receive a gift certificate worth \$10 for completing this study.

## **CONTACT**

If you have questions at any time about the study or the procedures, you may contact the researcher, Jamie Mayer, at the Department of Speech and Hearing Sciences, (812) 855-0666 in Bloomington or (608) 217-4073 in Madison, and [jfmayer@indiana.edu](mailto:jfmayer@indiana.edu).

If you feel you have not been treated according to the descriptions in this form, or your rights as a participant in research have been violated during the course of this project, you may contact the office for the Indiana University Bloomington Human Subjects Committee, Carmichael Center L03, 530 E. Kirkwood Ave., Bloomington, IN 47408, 812/855-3067, by e-mail at [iub\\_hsc@indiana.edu](mailto:iub_hsc@indiana.edu).

## **PARTICIPATION**

Your participation in this study is voluntary. That means that you can stop at any time and there is no penalty or cost for doing that. Also, your medical care and therapy will not be affected. If you decide not to participate, your data will be returned to you or destroyed.

## **CONSENT**

I have read this form and received a copy of it. I have received answers to the questions I have asked. I agree to take part in this study.

Subject's  
signature \_\_\_\_\_ Date \_\_\_\_\_

Witness  
signature \_\_\_\_\_ Date \_\_\_\_\_  
(required if form is read to subject)

Consent form date: 2/27/05

## Appendix B. N-back Instruction Script

### 0-Back

SHAPES TUTORIAL    **Materials:** 2-back banner, viewer

We are going to watch some shapes through a simulated television. Here are all of the different shapes (*Point to each shape and name*). I want you to look for all of the stars. (*Put banner through viewer so that first shape is showing*). Every time you see a star, I want you to say “match” out loud (*or point to the star, if subject cannot say it*).

FACES TUTORIAL    **Materials:** 0-back face pictures in folder

Now we are going to do the same thing with faces. I am going to show you some pictures of faces. I am going to show you a target face, then whenever you see that face I want you to say “match” out loud (*or point to it if subject cannot say it*). Good! Are you ready to try the short practice on the computer?

COMPUTER PRACTICE    **Materials:** 0-back practice script on computer

Now we are going to look at some faces on this computer screen (*point*). The faces will appear on the screen one at a time (*run program; target face will appear*). Here is your target face (*show target*). Whenever you see this picture on the screen, press the “?” key (*for right-handed subjects, “z” key for left-handed subjects or subjects with right upper extremity weakness*). When subject is ready, push the **space bar** to start the program: target face will disappear and program will run.

EXPERIMENT    **Materials:** 0-back scripts on computer

Now you will do the full task. It is the same as the practice, but longer. This time, your job is to pick out all of the pictures that look like this (*run program, target picture will appear first*). When you see the target picture, press the key as fast as you can and try not to make any mistakes. If you forget which picture you saw, just watch for the next picture to come up and try to remember that one. Are you ready? (*Push **space bar** for the target to disappear and program to begin*).

### 1-Back

SHAPES TUTORIAL    **Materials:** 1-back banner, viewer

This time you are going to look for the same shape in a row. So every time the shape you see is the same as the one before say “match” out loud (*or “point to the shape” if subjects cannot say it*). For example, if you see this shape first, and then you see this shape, that is a match. Now you try. (*Move banner through viewer until finished*).

FACES TUTORIAL    **Materials:** 1-back face pictures in folder

Now we are going to do the same thing with faces. Your job is to look for matching faces in a row. When the face you see matches the one you just saw, say “match” out loud (*or point to it, if subject cannot say it*).

**COMPUTER PRACTICE**      **Materials:** 1-back practice script on computer  
Now we'll do the same thing on the computer. In this practice, you're going to see some faces. Each face will appear on this computer screen one at a time. It is your job to look for matching faces in a row. When the face on the screen matches the face that you just saw, press the "?" key (*for right-handed subjects; "z" key for left-handed subjects or subjects with right upper extremity weakness*).

**EXPERIMENT**      **Materials:** 1-back script on computer (HI, lo, and neutral)  
Did the practice make sense? Good!  
Now you will do the full task. It is the same as the practice, but longer. Remember, when matching pictures appear in a row, push the key as fast as you can and try not to make mistakes. If you forget which picture you saw, just watch for the next picture to come up and try to remember that one. Are you ready?

## **2-Back**

**SHAPES TUTORIAL**      **Materials:** 2-back banner, viewer  
This time, I want you to watch for every other shape to match. So, whenever the shape you see is the same as the shape you saw two before, say "match" out loud (*or "point to the shape" if subject cannot say it*). (*Indicate an example of a 2-back*). Now I'm going to slide some shapes through here without saying anything. Remember, I want you to look for all of the times when the shape that you see is the same as the shape you saw 2 before.

**FACES TUTORIAL**      **Materials:** 2-back face pictures in folder  
This time I want you to say "match" (or point) when you see a face that is the same as one which was *two* before—so every *other* face.

**COMPUTER PRACTICE**      **Materials:** 2-back practice script on computer  
In this practice, you're going to see faces again on the computer screen, and each one will appear one at a time, just like before. This time, your job is to press the "?" key (or "Z") as fast as you can when you see a face that is the same as the face two before – so every *other* face. Here is your practice run. (*Run practice script*).

**EXPERIMENT**      **Materials:** 2-back script on computer (HI, lo, neutral)  
Did the practice make sense? Good!  
Now you will do the full task. It is the same as the practice, but longer. Remember, when every other picture matches, push the key as fast as you can and try not to make mistakes. If you forget which picture you saw, just watch for the next picture to come up and try to remember that one. Are you ready?

## **Jamie F. Mayer**

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### Research and Teaching Interests

Adult Neurogenic Cognitive-Communication Disorders  
Interactions between domain-specific and domain-general faculties in the generation, assessment, and treatment of neurogenic cognitive-communicative disorders  
Cognitive Neuroscience  
Cognitive Psychology

### Education

**Ph.D.**, Speech and Hearing Sciences, Indiana University, Bloomington (Feb. 2007)  
Minor: Neural Science.  
Dissertation: The nature of working memory deficits in aphasia.  
Advisor and Chair: Laura Murray, Ph.D.

**M.A.**, Speech and Hearing Sciences, Indiana University (May 2000)

**B.A., Highest Distinction**, Speech and Hearing Sciences, Indiana University (May 1998). Minors: Psychology, Spanish.

### Publications in Preparation

*Mayer, J. F.* (in preparation). Exploring the nature of working memory in aphasia.

*Mayer, J. F.* (in preparation). Critical review: Conceptualizing working memory and aphasia.

*Mayer, J. F., Murray, L. L., & Karcher, L. A.* (in preparation). Naming treatment in aphasia: An investigation of the role of stimulus complexity.

### Publications

*Mayer, J. F.* (2004). [Review of the book The big book of concepts]. Journal of Child Language, 31, 247-253.

*Mayer, J. F.* (2003). The role of fMRI in imaging untreated and spontaneous recovery in aphasia: Interface between technology, theory, and clinical care.

Perspectives on Neurophysiology and Neurogenic Speech and Language Disorders  
(American Speech-Language-Hearing Association Division 2), 13 (3), 4-7.

*Mayer, J. F., & Murray, L. L.* (2003). Functional measures of naming in aphasia: Word-retrieval in confrontation naming versus connected speech. Aphasiology, 17 (5), 481-498.

*Mayer, J. F., & Murray, L. L.* (2002). Treatment of alexia in chronic aphasia. Aphasiology, 16 (7), 727-743.

Presentations

*Mayer, J. F., Murray, L. L., & Turkstra, L. S.* The nature of working memory deficits in aphasia. Seminar presented at: American Speech-Language Hearing Association, Miami, FL, November 18, 2006.

*Murray, L. L., Rey, O., Kean, J. A., & Mayer, J. F.* Sentence processing in aphasia: Dual-task and sentence-type effects. Poster presented at: American Speech-Language Hearing Association, Miami, FL, November 16, 2006.

*Mayer, J. F., Murray, L. L., Ikuta, T., Kean, J. A., & Rey, O.* Verbal fluency in aphasia and right hemisphere damage: Qualitative analysis. Presented at: American Speech-Language Hearing Association, San Diego, CA, November 18, 2005.

*Mayer, J. F., Murray, L. L., Ikuta, T., Kean, J. A., & Rey, O.* Verbal fluency in aphasia and right-hemisphere damage: Qualitative analysis reveals relationship to cognitive factors. Presented at: Clinical Aphasiology Conference, Sanibel Island, Florida, June 2, 2005.

*Murray, L. L., Mayer, J. F., Kean, J. A., Rey, O., & Ikuta, T.* Verbal fluency in aphasia and right-hemisphere damage: The effects of dual-task conditions. Presented at: Clinical Aphasiology Conference, Sanibel Island, Florida, June 2, 2005.

*Mayer, J. F., Murray, L. L., & Karcher, L. A.* Naming treatment in aphasia: An investigation of the role of stimulus complexity. Presented at: Clinical Aphasiology Conference, Park City, Utah, May 27, 2004.

*Mayer, J. F., Murray, L. L., & Karcher, L. A.* Treatment of anomia in severe aphasia: What matters? Presented at: American Speech-Language Hearing Association, Chicago, IL, Nov. 13, 2003.

*Mayer, J. F., & Murray, L. L.* Quantifying word retrieval in aphasic discourse: Validity and feasibility. Presented at: International Neuropsychological Society Annual Meeting, Honolulu, Hawaii, Feb. 8, 2003.

*Mayer, J. F., & Murray, L. L.* Functional measures of naming in aphasia. Presented at: American Speech, Language, and Hearing Association Annual Convention, Atlanta, GA, Nov. 23, 2002.

*Mayer, J. F., & Murray, L. L.* Functional measures of naming in aphasia: Word retrieval in confrontation naming versus connected speech. Presented at: Clinical Aphasiology Conference, Ridgedale, MO, June 4, 2002.

*Mayer, J. F., & Murray, L. L.* Treatment of alexia in chronic aphasia. Presented at: American Speech-Language Hearing Association Annual Convention, Washington, DC, Nov. 19, 2000.

*Mayer, J. F., & Murray, L. L. A comparison of reading-specific and general cognitive approaches to treating alexia in chronic aphasia. Presented at: Clinical Aphasiology Conference, Waikoloa Beach, Hawaii, May 28, 2000.*

### Selected Honors & Awards

- 2005 American Speech-Language Hearing Foundation New Century Scholars Doctoral Scholarship
- 2005 Bernice Eastwood Covalt Memorial Scholarship, Indiana University
- 2005 College of Arts and Sciences Dissertation Research Fellowship, Indiana University
- 2005 Clinical Aphasiology NIDCD Student Fellow Award
- 2004 Clinical Aphasiology NIDCD Student Fellow Award
- 2003 American Speech-Language Hearing Association Student Research Travel Award
- 2002 American Speech-Language Hearing Foundation Graduate Student Scholarship
- 2001 Chancellor's Fellowship, Indiana University (2001-2003)
- 1999 Speech and Hearing Foundation Exchange Club Scholarship (1996-1999)
- 1999 Audrey Heller Continuing Education Award
- 1998 Graduate Assistantship Award, Indiana University (1998-1999)
- 1998 Alpha Kappa Alpha Educational Achievement Scholarship
- 1998 Senior Achievement Award, Indiana University
- 1998 Norvelle Undergraduate Achievement Award, Indiana University
- 1997 Phi Beta Kappa

### Professional Affiliations

Certified Member, American Speech, Language, and Hearing Association  
Division 2: Neurophysiology and Neurogenic Speech and Language Disorders  
CCC-SLP: Certificate of Clinical Competence- Speech Language Pathology  
Associate Member, International Neuropsychological Society  
Associate Member, Academy of Neurologic Communication Disorders and Sciences

### Professional Experience

#### ***Appointments:***

Visiting Assistant Professor, Northern Illinois University, August 2006-May 2007

Courses taught:

- COMD 323 Anatomy and Physiology of the Speech and Hearing Mechanism
- COMD 574 Cognitive-Linguistic Disorders of Neurologically Impaired Adults
- COMD 429 Assessment Procedures in Communicative Disorders
- COMD 425 Neuropathologies of Speech and Language

Clinical activities:

- Initiated and co-developed local, student-led stroke support group

**Research:**

Research/Teaching Assistant, September 2003-June 2004

Cognition and Language lab, University of Wisconsin-Madison

Supervisor: Lyn S. Turkstra, Ph.D., Associate Professor

Research/Teaching Assistant, August 2001-August 2003

Adult Cognition and Communication lab, Indiana University

Supervisor: Laura L. Murray, Ph.D., Associate Professor

Research/Teaching Assistant, August 1998-December 1999

Master's Level Student Academic Appointment, Indiana University

Supervisors: Barbara Fazio, Ph.D., Assistant Professor

Rita Naremore, Ph.D., Professor

**Teaching:**

- Guest Lectures: University of Wisconsin-Madison
  - Spring 2006: PM&R Resident Didactics
  - Fall 2005: CD 712 Dysphasia in Adults
  - Spring 2005: CD 712 Dysphasia in Adults, CD 942 Seminar—Language Pathology, Adults
  - Spring 2004: CD210 Speech and Language Function in the Brain, CD942 Seminar—Language Pathology, Adults
- Clinical Instructor: Western Michigan University
  - Summer 2003: SPPA 670 Clinical Practicum
- Instructor: Indiana University
  - Spring 2002: S307 Cognitive and Communicative Aspects of Aging

**Clinical:**

- *Populations served: Children (birth through school-aged), developmentally disabled adults, and adults with acquired, neurogenic communicative and swallowing disorders.*
- *Settings: Acute medical, acute rehabilitation, sub-acute rehabilitation, home care, and outpatient.*

Certified Speech-Language Pathologist, August 2003-May 2006

University of Wisconsin Hospital and Clinics, Madison, WI

Supervisor: Vicki Hill, CCC-SLP

Certified Speech-Language Pathologist, June 2002- September 2002

The Springs at Bronson Place, Kalamazoo, MI

Supervisor: Melony Bishop, OTR

Certified Speech-Language Pathologist, May 2000- May 2002

Riverview Hospital Rehabilitative Services, Noblesville, IN

Supervisor: Susan Holbert, CCC-SLP

Clinical Externship, March 2000- May 2000

Veteran's Administration Medical Center, Indianapolis, IN

Supervisor: Susan McGarvey-Toler, CCC-SLP

Clinical Externship, January 2000- March 2000

Washington Township Schools, Indianapolis, IN

Supervisor: Mary Auberry, SLP